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ANNUAL DISCOURSE

THE INVOLUNTARY PLIGHT OF THE VOLUNTARY HOSPITAL*

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BOSTON

IN his autobiography, Benjamin Franklin wrote: "In 1751, Dr. Thomas Bond, a particular friend of mine, conceived the idea of establishing a hospital in Philadelphia . . . for the reception and cure of poor sick persons, whether inhabitants of the province or strangers." Thus was born the Pennsylvania Hospital, the first voluntary hospital in the United States. The seal chosen for the institution "bore a device of the Good Samaritan conveying the sick man to an inn, with the inscription 'take care of him, and I will repay thee.'"

Those who contributed financially to this enterprise thereupon elected a board of managers. The Board not only chose the physicians who would practice in the new hospital, but passed on their "performance as practitioners." Though physicians were elected for terms of one year, the first three physicians "offered to serve the hospital for three years without compensation and to supply all the medicines for that time at their own expense." It is significant that when Dr. Thomas Bond was appointed to the medical staff, he resigned from the Board of Managers, on which he had served during the hospital's first year.[1] This hospital therefore represents the oldest incorporated and surviving voluntary hospital in the United States and the establishment of principles that have more or less guided all future nonprofit hospitals.

From this humble beginning has developed a gigantic hospital system, available to the rich as well as the poor. Today, hospitals are the sixth largest industry in the nation, and Massachusetts has more than its share of this industry. Facts and figures furnished by the Massachusetts Hospital Association reveal that there are 218 hospitals in Massachusetts. Of these, 125 — more than half — are nonprofit. The voluntary hospitals are distributed as shown in Table 1.

Since the preponderance of the population is in eastern Massachusetts and Boston itself is a renowned medical center, it is not surprising that half the hospitals and two thirds of the hospital beds are in Greater Boston. These hospitals employ altogether nearly 26,000 persons — more than 2 employees for every hospitalized patient.

The total assets of all hospitals in Massachusetts exceed $500,000,000; of this, $310,000,000 is in voluntary hospitals alone. The total annual hospital expense is almost $330,000,000, of which the share of the nonprofit hospitals is $100,000,000. But the most significant and disturbing fact of all is that the voluntary hospitals are now spending annually $3,000,000 more than their total income.

As far as the patient is concerned, the cost of hospitalization has risen almost to the point of diminishing returns. At the same time, hospitals are finding it necessary to reduce their capital assets to meet expenditures. It is evident that this situation cannot be permitted to continue too long.

The deteriorating financial situation in hospitals has been variously attributed to the economic depression of the 1930's, World War II, inflation and high taxes. The war forced hospitals to compete with high wages in industry. Before the war, hospital employees worked at standard salaries lower than those in other fields. Whereas wages then were only 40 per cent of hospital expenditures, today the figure is 60 to 65 per cent. In addition, many expensive new drugs have appeared, and the cost of food, surgical supplies and equipment has skyrocketed. Income from invested funds — Government subsidy, Community Chest, gifts and contributions — accounted for 11 per cent of total hospital income in 1952, as compared with 29 per cent in 1935. Since World War II, hospital charges have risen 161 per cent, in contrast to physicians' fees, which have gone up 45 per cent. Governing boards, which did such a superb job in building the nation's hospitals, have virtually ceased to be an adequate source of contributions.

Economies are, of course, being attempted. Hos-

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pitals have called in consultants to help simplify the operations of the various departments. Some nurses' training schools have been abolished. There is more group purchasing. Greater economies are undoubtedly possible. Are physicians perhaps ordering unnecessary laboratory procedures and wasting supplies? Are they discontinuing expensive drugs as soon as these drugs are no longer needed? Unfortunately, savings are possible only in the 35 to 40 per cent expended for supplies and upkeep. Salaries and wages, which account for the remainder of the expense dollar, cannot be reduced. With more than 1300 jobs unfilled, it is obvious that hospital employees are not overpaid.

Possibly, something can be learned from the management of proprietary hospitals. Generally speaking, their rates are about the same as those of the nonprofit hospitals, and patients are pleased with the services. Although proprietary hospitals are subject to taxation, I have been told by owners that they operate at a profit. To be sure, most of the patients have common ailments, and pay full rates. There is no teaching or research. On the other hand, should patients in voluntary hospitals be forced to underwrite prestige activities?

TABLE 1. *Distribution of Voluntary Hospitals in Massachusetts.*

LOCATION	HOSPITALS	NO. OF BEDS	AVERAGE CENSUS	OCCUPANCY
				%
Boston	26	5,360	3,872	72
Metropolitan Boston	36	4,583	3,392	74
Remainder of State	63	6,430	4,754	74
Totals	125	16,373	12,018	

Principles of Payment for Hospital Care, a pamphlet published by the American Hospital Association (September 1953, page 8) declares that "Major expenditures . . . for medical research purposes . . . should . . . be financed from sources other than patients being served in a particular hospital."

Regarding teaching, the pamphlet comments: "Ideally, the cost of educating and training . . . should be financed by the whole community through a combination of public resources and private contributions, rather than by the sick patient representing a small percentage of the community, who is usually in the poorest position to meet such cost."

In consequence of all these developments, voluntary hospitals throughout the country are looking for new sources of income. Some apparently have been influenced by the propaganda that doctors make a great deal of money in hospitals and so should help meet hospital losses. However, both the American Medical Association and the American Hospital Association believe that the physician's role in hospital support should be that of any other citizen of like means.

Nevertheless, some hospitals have adopted plans for compelling staff physicians to pay for annual deficits incurred by the hospitals. One hospital "requested" staff members to make $1,000 "donations" to cover operating deficits. Another set up a compulsory plan for the collection of professional fees, the hospital to keep 50 per cent of the amount collected. Some hospitals require initiation fees of $150 to $500 for the privilege of becoming a staff member. One hospital was charged with placing the various services on the block — accepting bids for exclusive staff privileges. Some hospitals require the physician to pay for each patient admitted. Some employ full-time anesthesiologists, pathologists and roentgenologists, and make a profit on their services, even though this arrangement is contrary to law and the ethical principles laid down by the American Medical Association. Schemes such as legalizing bingo, lotteries like the ones operated in Mexico and annual sweepstakes like those held in Ireland have also been considered on the assumption that as long as people like to gamble, the hospitals, too, might as well benefit. For example, in a *History of the Massachusetts General Hospital* by Nathaniel Bowditch the following item appears (page 53): "June 6 (1820) — The trustees declined applying to the legislature in aid of a project for a lottery, a portion of the profits of which were to be for the use of the hospital."

For years, voluntary hospitals in Massachusetts had partly subsidized industry and welfare departments by hospitalizing Workmen's Compensation and welfare cases at rates below those charged to the general public for comparable services. On December 1, 1948, however, insurance companies began paying hospitals on the basis of actual cost or what is charged to the general public, whichever is lower. As of January 1, 1955, hospitals are to be reimbursed on the same basis for the care of welfare cases.

The purchase of hospital services by agencies acting as a third party has necessitated improvements in hospital accounting. Since voluntary hospitals do not pay income taxes, their accounting methods have left something to be desired. Dr. Robert S. Myers,[2] assistant director of the American College of Surgeons, wrote: "It would seem that hospitals are the only major business in which unreliable statistics are thoughtlessly selected, laboriously collected, promiscuously dissected, and unreservedly accepted as facts which accurately gauge achievements." In Massachusetts a new division of hospital costs and finances, in the Commission on Administration and Finance, has been in operation since January 1, 1954. Under the penalty provisions of Chapter 636 of the *Acts* of 1953, a hospital may be fined $500 for failure to provide such cost information as may be required. As a result hospitals are on the way to having a uniform system of accounting and cost analysis.

The Health Insurance Council reported that as of mid-November, 1954, 103,000,000 persons (approxi-

mately 62 per cent of the total population of the United States) had voluntary health insurance against hospital expenses. This includes more than 70 per cent (3,317,000) of the population in Massachusetts. Despite this encouraging fact there are still many patients who are unable to pay full hospital costs. Although most, if not all, of the nonprofit hospitals have funds earmarked to assist indigent patients admitted to their respective institutions, this money is no longer enough. Additional financial assistance is necessary. One such source is the Red Feather services. In the past, so far as the Boston Metropolitan Area was concerned, fund payments were based on operating deficits. Since 1951, however, payments have been limited to units of free service given to patients. The amounts paid to some of the hospitals in Metropolitan Boston, together with related information, are listed in Table 2.

Staff-Trustee Relations

Jerome Preston,[3] president of the Board of Trustees of the Massachusetts Memorial Hospitals, has made the following comment on this most vital aspect of hospital administration:

> A hospital is unique among institutions in that staff doctors are independent of it and have no responsibility for its finances. Their professional responsibility toward the patient, like the financial responsibility of the trustee toward the donor, cannot be delegated. Such a division of responsibility is an organizational monstrosity, one that has troubled both doctors and trustees for years.

In addition, hospital income now comes largely from the staff's private patients. This combination of factors has created a definite impairment of staff-trustee relations.

The House of Delegates of the American Medical Association has taken cognizance of this schism. In December, 1951, it approved a report pointing out that "Since the physician and hospital are interdependent, it is incumbent on both to be interested in all phases of their scientific and financial relationship."[4]

Eighteen months later the House of Delegates approved another report that commended the people who serve on governing boards and added:

> They are not necessarily particularly informed as to the many intricate problems involved in the production of good medical care. . . . If they have opinions on critical matters [these opinions] are likely to be gained from the personal advisor or by casual contacts at social gatherings or on the golf course.

Various methods were recommended by which the medical staff could have "free and direct access to the governing board." Of special interest is the recommendation that members of the medical staff serve on the governing board.[5]

Regarding this recommendation, the *Detroit Medical News*, on October 26, 1953, made the following editorial comment:

> An agreement to accept physicians as members of Boards of Trustees of hospitals has been reached. . . . The formation of a partnership by the staff of a hospital and its trustees . . . augurs well.

In the opinion of Dr. Malcolm T. MacEachern, director of professional relations of the American Hospital Association, the policy of having the medical staff represented on the governing body is not in accordance with the principles advocated by the American Hospital Association. Some of the reasons

Table 2. *Amounts Paid to Hospitals.*

Hospital	Total Expenditures*	Free Care (Indigents & Public Agency)	United Community Services Payments	Uncollected Accounts†
	$	$	$	$
Massachusetts General	9,733,543	992,831	249,550	277,830
Children's	3,695,395	244,073	56,806	29,767
Massachusetts Memorial	2,993,238	489,943	198,100	33,220
Peter B. Brigham	2,804,466	242,915	117,470	51,448
New England Deaconess	2,637,793	101,735	11,384	50,909
Boston Lying-in	1,644,990	17,690	5,123	5,888
Massachusetts Eye & Ear Infirmary	1,394,945	135,660	55,436	13,253
Carney	1,256,196	88,693	23,350	2,614
New England	927,261	15,318	8,103	25,220
Free Hospital for Women	729,673	188,751	10,326	5,845
Robert B. Brigham	607,948	77,669	33,450	1,204
Boston Floating	401,605	52,624	28,572	5,584
Infants	293,114	39,282	21,647	73,048
Mount Auburn	1,832,872	58,297	19,047	27,271
Newton-Wellesley	1,808,604	98,500	71,000	23,727
Malden	1,146,054	40,021	17,194	22,274
Waltham	1,023,114	40,781	6,476	3,778
South Shore	842,979	2,910	1,000	7,824
Whidden	584,601	6,561	4,711	1,504
Choate	397,583	5,641	3,480	5,602

*From the United Community Services report for 1954.
†According to American Hospital Association uncollectable income varies from 4 to 7 per cent, depending on size of hospital: the larger the hospital, the smaller the percentage.

advanced are that membership on the governing board gives undue publicity to the individual physician; that he may use his position to promote himself on the staff; that the staff may come to regard him in the light of an inspector; that he may exert his authority in the employment of hospital personnel; and that finances and business are foreign to him.[6]

Granting that all objections to having physicians on governing boards are valid, how much better qualified are the laymen to serve as hospital trustees? In 1952, Raymond P. Sloan, for many years a hospital trustee and editor of *The Modern Hospital*, wrote a book, *This Business of Ours*. The book, described as "A Guide Book for Hospital Trustees, Hospital Workers and Laymen," has this to say:

> Although some parallel can fairly be drawn between hospital operations and that of big business, there is a

> point at which marked divergencies will be noted. The industrial organization is governed by those who supposedly know their fields; the hospital organization is governed by men and women who have little, if any, knowledge of its professional affairs. . . . [The doctor] sees a layman possessing little acquaintance with medical science or hospital technique . . . placed in a position where he dictates policies and exerts controls over those who have spent years in acquiring professional knowledge. What could be more inconsistent?

The director of the Doctors Hospital in Seattle (Dr. Robert F. Brown) believes that more is to be gained than lost by having physicians serve as trustees. In the opinion of many hospital administrators, according to Dr. Brown, physician trustees foster better understanding between the medical staff and the board. Although nonmedical boards become more easily a rubber stamp in approving candidates for staff appointment, physician trustees are more inclined to evaluate applicants on their credentials. He refutes the criticism that physicians are lacking in business ability by pointing out that they serve as trustees of universities and research foundations, on the corporate boards of industry and in the insurance field.[7]

Dr. Frank H. Lahey, in his presidential address before the New England Surgical Society on September 10, 1932, spoke as follows:

> I believe that on every hospital staff there should be several members of the staff who are trustees of the hospital; . . . if the members of the medical profession do not assert an interest in and accept a responsibility for their economic management, they, the medical men, will be pushed farther and farther down the scale until they assume the position from which they are now not far distant, of "hired men," more or less — under the power and control of trustees and superintendents.

The set-up of the Massachusetts Blue Shield Corporation could well be used as a pattern for hospital governing boards. Of Blue Shield's members, one is a banker, 9 represent labor and industry, and 5, or a third, are physicians. Thus, Blue Shield has broad public *and* medical representation. On hospital governing boards at least 3 physician trustees, with staggered terms, should be *elected* by the staff. It would be well, too, if these physicians were not permitted to succeed themselves. This system would over the years permit a large percentage of the staff to serve on the governing board and to become better informed of trustees' problems. It would also implement the recommendation of the House of Delegates of the American Medical Association that "every professional man on the appointed staff have a voice in the professional management of the institution."[4]

Regional Integration

Another aspect of the problem facing the voluntary hospital that deserves serious consideration is regional integration. In a way, voluntary hospitals are engaged in a highly competitive enterprise, even though they are erected in the public interest, are exempt from taxation, receive funds from philanthropic-minded individuals and agencies, and may even be assisted by federal and other tax funds. They are in competition not only with hospitals entirely supported by taxation but also with one another. They compete for funds; medical, nursing, and technical personnel; prestige; and even for patients. Since all nonprofit hospitals are dependent on the beneficence of the public, their attitude toward one another should be co-operative rather than competitive. This competition exists not only among hospitals located in a single city but also between urban and suburban hospitals. Increased occupancy of community hospitals results in decreased occupancy of city institutions.

What the Hoover Commission had to say about the various federal medical systems can well be applied to voluntary hospitals: "They go their own ways, make their own plans, build, staff, and run hospitals and clinics with little knowledge of and no regard for the operation of others." Voluntary hospitals, finding themselves with a high average census, have embarked on building programs even though there are empty beds in nearby institutions. Under the Hospital Survey and Construction Act, construction costs in Massachusetts during the past five years have averaged over $17,500 a bed. If hospital needs were considered on a regional rather than on an individual basis, overlapping facilities would be avoided, and substantial savings would result. Less busy hospitals would benefit, for the more closely the average occupancy of a hospital approaches 100 per cent, the lower the unit of cost of medical care.

One may ask how effective integration can be accomplished. Here are a number of recommendations by the American Hospital Association Commission on Financing of Hospital Care: regular meetings for discussion of common problems; joint recruitment of nurses; sharing of skilled personnel; joint purchasing and joint collections; joint reporting of operating data; agreement to treat certain types of cases and to provide particular services in specified hospitals; and merger of hospitals when it is in the interest of effectiveness and economy.

Since most, if not all, of the hospitals in Greater Boston belong to the Boston Hospital Council, this agency could well serve as a medium for discussion of mutual problems in this area. Similar councils, to operate in the same manner, could be organized in other sections of Massachusetts. Due consideration should be given, of course, to overlapping regions. Possibly, the Massachusetts Hospital Association could undertake the over-all job of founding these regional councils.

The United Community Services of the Metropolitan Boston Area has become interested in this problem. It has obtained a grant of $125,000 from the United States Public Health Service for a three-year basic research project to evaluate improvements that may result from regional co-ordination in an urban area.

To be truly effective, the integration of hospitals

within an area, as I see it, would require an interchange between co-operating hospitals of courtesy-staff privileges for the care of private patients. Before integration, hospitals should be approved by the Joint Committee on Accreditation. In this way, the qualifications of staff members would meet the standards of all co-operating hospitals. No change should be made in the *closed-staff setup* for the care of service cases. I might observe at this point that integration has been tried in some communities and has been found to work very well.

With integration and reciprocal staff relations in effect, patients will be able to have free choice of physician. At present they can only be sure of "free choice" when services are rendered at home or in the office, for, if the patient insists on a hospital where his physician is not a staff member, he must give up either his physician or the hospital of his own choice.

A clergyman made the following observation:

> And speaking of hospitals here is another source of misunderstandings and ill will. The doctor wants to head off socialized medicine. One of the arguments he uses is that the patient should be free to choose his physician. . . . But often the doctor does not practice what he preaches and [does not allow] his patient freedom to choose the hospital he wants to enter, but insists — to the point of breaking the patient-doctor relationship — on the hospital of the doctor's choice.[8]

Unfortunately, the minister quoted appears to have overlooked the *doctor's* dilemma — that sometimes the hospital of the patient's choice is one in which the doctor does not have staff privileges!

Much more to the point is an editorial in the *Bulletin of the Columbus* (Ohio) *Academy of Medicine* (March, 1953) proposing that every member of the Academy be given courtesy-staff membership in every hospital in Columbus. It asks a pertinent question:

> By what specious reasoning do we decide that a certain doctor can use the facilities of some hospitals, but not others? Such decisions are apparently based less on professional considerations than . . . on the whims or personal prejudices of those entrusted with the decisions.

Under the present system, the staff physician's freedom of choice is also restricted. He may have as consultants only members of the staff of the particular hospital to which his patient has been admitted. With integration of hospitals on a regional basis and interchange of staff privileges, both physicians and patients will be better served. Physicians will have unrestricted free choice of consultants and patients will achieve free choice of physician and free choice of hospital.

With regional integration some hospitals could be reserved for routine cases. These hospitals would not have to be so elaborately equipped and staffed as institutions reserved for patients who require more intensive study and more complicated procedures. There would thus be less duplication of services and equipment. Since fewer technicians would be necessary, there would be a saving in salaries.

If a patient in one hospital required the facilities of a more elaborately equipped hospital, he could be transferred there and yet remain under the care of the original physician. Last but not least, the *patient* would save. The patient with a routine illness would no longer be charged for a share of the cost and technical servicing of equipment rarely used.

The larger, better known and busier hospitals that are not interested in pooling their facilities with more modest institutions should bear in mind the value of integration in times of disaster such as fire or war. Any voluntary hospital contributing to the difficulties of other voluntary hospitals may be contributing to the breakdown of the whole voluntary system.

When there is actually a need for new hospital construction, consideration should be given to institutions in which some floors are used as a hospital and other floors as a hotel. Such an arrangement would shorten the stay of the patient in the hospital section and reduce the costs of hospitalization.

Another method of cutting the high costs of hospital construction and equipment would be to increase the use of existing facilities. It has been pointed out that the elimination of week ends and holidays would increase hospital capacity by about a third.[9]

Virtually the same thing was said by the president of the American Medical Association[10] who suggested that "making hospital services available on a full week or at least a six-day basis, rather than the present four-and-a-half to five-day utilization, would not only reduce the cost of the care of the individual but would lessen the number of hospital beds needed to service a community."

Another physician,[11] this time a patient, wrote as follows:

> At 4:30 P.M. on Friday I was taken to the hospital, presumably with a kidney stone. X-rays were in order, of course, and some kidney function tests.
>
> But the laboratory operates on a five-day week. From 4 P.M. Friday to 8 A.M. Monday, nobody is there . . . So I just lay around all week-end, waiting for the technicians to come back.
>
> It's obvious that the forty-hour week in a hospital is as much a bane to patients as it's a boon to employes. . . .
>
> The hospital keeps its switchboard going twenty-four hours a day, seven days a week. I suggest that it could keep *all* departments open on Saturday and Sunday, 8 A.M. to 4 P.M. Even though this would cost more in overtime salaries, the increment would not be a very big item in the total budget. And the returns would be tremendous.

Look magazine (August 24, 1954) took note of one ingenious attempt to solve this problem of the "lost week end" in hospitals: "Week-end patients," the news item read, "are being welcomed at San Francisco's Mt. Zion Hospital. To take up Saturday and Sunday slack, the hospital is seeking patients from among overworked folk who need a check-up and could use two days' rest."

Finally, the Committee on Financing Hospital Care warned that "Overbuilding, in the attendant failure to make full use of bed capacity and diagnostic and therapeutic facilities, should be avoided." It recom-

mended "reducing seasonal and week-end lulls whenever practicable, and making diagnostic services available at all times." It also advised programs for making preventive medical care available at a reasonable cost to all groups in the population.

Audits

The desirability of self-audits by hospital trustees merits consideration.

The Joint Committee on Accreditation of Hospitals requires that the quality of professional work in a hospital be under constant examination by the staff. Meetings must be held at least monthly for the purpose of reviewing the medical care of patients within the hospital.

It seems only proper that the work of the governing board should be subjected to a similar self-audit and individual members made answerable for their functions just as physicians are for the care of patients. Here are some pertinent questions that might be asked at one of these governing-board audits: Are the trustees receiving sound and unbiased advice from impartial physicians? Is the work of the administrator being adequately supervised? Are there lapses in hospital-physician and in hospital-public relations? How successful are the money-raising activities? How well are individual trustees attending board meetings? How well are they carrying out their committee assignments?

When Benjamin Franklin was unable to get the board members of the Pennsylvania Hospital to meetings on schedule, he had the following resolution passed: "[that] each member is to pay two shillings sixpence for total absence and one shilling for not coming on time, and for every hour's absence after the fixed time, sixpence per hour, all of which fines to be disposed of as the majority may direct. The town clock, or should that not strike, the watch of the oldest person present to be the standard for determining the time."[1]

The governing board, like the medical staff, should make every effort to disclose its own weaknesses and errors, and to correct them. If anything, the work of the trustees should be checked even more carefully than the work of the professional staff. Physicians, at least, are trained for their jobs, whereas the individual lay trustee assumes a responsibility for the conduct of a public utility about which he usually knows very little. So much for the trustees. May they find these suggestions helpful!

New Era in Hospital-Physician Relations

In June, 1950, the House of Delegates of the American Medical Association recommended that committees on hospital and professional relations be created on state and county levels. These committees were to receive complaints from any physician, hospital, medical organization or other interested person or group about professional or economic relations existing between physicians and hospitals.[12] Such committees were appointed by the Massachusetts Medical Society and the district medical societies.

A further step in the improvement of hospital-physician relations was taken a year ago by the Council of the Massachusetts Medical Society and the trustees of the Massachusetts Hospital Association. In short, the two organizations agreed that no governing board should deprive a physician of staff membership or reappointment, or change his privileges in any way without informing him of the charges against him and without previous consideration by the staff; that on request a review should be made by a committee with equal representation from the staff and governing board; that if this committee was unable to make a majority recommendation, or if any of the parties involved so desired, a request for an opinion should be made to the presidents of the Society and the Association; that, on receipt of such request, the president of the Society should appoint 3 members of the Society, and the president of the Association 1 hospital administrator and 2 hospital trustees to act as an appeal board; that the 6 appointed members should select a lay chairman; and that the findings of the various committees should be transmitted to all the parties involved.

The *Norfolk Medical News*, in a subsequent editorial (November, 1954), reported as follows:

> As a result of this cooperative action between the M.M.S. and M.H.A., staff members who feel they have been unjustly treated now have a court of appeal. These procedures, of course, carry no legal force, and are not binding on the governing boards of hospitals. However, the moral effect on hospitals belonging to the M.H.A. (and practically all belong) will be to induce these institutions to abide by the recommendations.

Future

The most vital question at present is the outlook for the voluntary hospital. In *The Hospital in Contemporary Life* (1949) Dr. Nathaniel W. Faxon wrote as follows: "What shall we do now? Confronted with the necessity of meeting hospital costs — and they must be met, some people are suggesting the common solution, turning it over to the government."

Evidence of the extent to which the federal Government has gone to the assistance of the voluntary-hospital system appears in the report of Dr. A. Daniel Rubenstein.[13] Since 1948, under Public Law 725, $13,990,190 has been allocated to 58 projects in Massachusetts for hospital and health-center construction. Of this, 79 per cent ($11,052,250) went to general hospitals, including teaching hospitals, maternity hospitals and laboratories; 3671 beds and 602 bassinets were added.

Fortunately for the voluntary-hospital system, "there were," in Dr. Rubenstein's[13] words, "no federal controls. Hospitals aided under the program have maintained absolute freedom of action." But if gov-

ernmental aid continues to be accepted, will the freedom of physician and hospital remain unimpaired?

Some hospital administrators, faced with continuing deficits, would have little or no hesitancy in placing their institutions, as Dr. T. F. Laye[14] puts it, "in a government socialistic noose." But it is fair to say that this practice would not meet with the overwhelming endorsement of the medical profession.

A somewhat gloomy view of the future of the voluntary hospital has been taken by the trustees of the Massachusetts General Hospital, which, in its 1953 report (pages 42-43), observed that "For the first time since 1936 the overall operations of the hospital did not end in a deficit. There is nothing in the present outlook to indicate that deficit years will be less frequent in the future than in the past."

On the other hand, MacEachern[15] appears to be optimistic regarding the future of the voluntary-hospital system. "I feel," he says "[that] the voluntary hospital is here to stay. It tends more and more to give higher standards of service, eliminates 'routinism' and retains certain humanitarian characteristics, more so than a government institution."

In general, however, the outlook for the community hospital appears to be better than that of the metropolitan hospital. Factors favoring the community institution are its convenience, civic pride and the availability of well trained men who have settled in the community. Some local hospitals are even being modeled after the university hospital. Also, since the community hospital may be the only hospital in its area, it receives from the public as well as from the staff physicians proportionally greater support than urban hospitals. Last year the Beverly Hospital put on a campaign. As of December 30, 1954, $625,000 had been subscribed by the public. In Fitchburg, $1,192,000 was raised by public subscription. The 48 members of the staff alone contributed $115,000. Of more than $600,000 raised by the Malden Hospital, exclusive of an additional single donation of $100,000, 96 physicians contributed $105,000, and of the $428,000 contributed by the public to the South Shore Hospital, $75,000 came from the staff.

Other straws in the wind indicate that the voluntary hospital promises to continue more or less unfettered for some time to come. On Long Island, New York, there is a labor-management coalition, formed to aid voluntary hospitals. At the end of four years the fund had reached $1,000,000. Last year, Indianapolis refused federal hospital funds but raised $12,000,000 locally.[16]

In this connection I cannot help observing once again that the circumstances attending the founding of the Pennsylvania Hospital seem peculiarly apropos to the problems that the voluntary hospital faces today. Franklin writes as follows in his *Autobiography:*

> The proposal [i.e., the founding of a voluntary hospital] being a novelty in America, and at first not well understood [Dr. Thomas Bond] met with but little success . . . I inquired into the nature and the utility of the scheme, and receiving a very satisfactory explanation, I not only subscribed to it myself, but enlarged heartily in the design of procuring subscriptions from others. Previously, however, to the solicitation, I prepared the minds of the people by writing on the subject in the newspapers, which was my usual custom in such cases, but which he had omitted.

Since governmental assistance was also wanted, and the Assembly appeared not too enthusiastic about the idea of a hospital, Franklin proposed a petition to the Assembly for public aid and devised a scheme for matching contributions by the Government and the public. In Franklin's words: "This condition carried the bill through."

It will be recalled that the first physicians of the Pennsylvania Hospital agreed to donate the necessary medicines in addition to their services. But "their generosity was not called on for long as the Managers soon undertook this financial burden." In the hospital minutes of December, 1752, the following note appears: Agreed that the Managers, each of them in Turn, solicit Subscriptions from the rich Widows and other Single Women in Town, in order to raise a Fund to pay for the Drugs."

A benefit performance given in 1759 cleared more than £47 for the hospital. In 1764 an eloquent minister preached a benefit sermon that yielded over £174 including a personal contribution of £5 from the orator. Charity boxes were distributed in various places, and by 1846 had yielded $19,093.44.[1]

Over the years, voluntary hospitals have not only continued to use the money-raising technics of the Pennsylvania Hospital, but have improved upon them. And yet the problem of hospital financing is still serious.

The Commission on Financing Hospital Care has acknowledged that voluntary prepayment is the backbone of hospital income, and that the financial stability of the voluntary hospital is increasingly dependent on this source of income. But the Commission believes that a danger is inherent in prepayment — that "it may encourage unnecessary utilization of hospital services and weaken or remove incentives to minimizing hospital expenditures." This, in turn, would increase the cost of such insurance to the subscriber. The Commission recommended:

1. Continuing programs of education to make the public more aware of its need to budget for hospital care by the purchase of prepayment insurance.
2. Encouragement of more employer participation in the cost of prepaid protection for employees and their families.
3. The development of methods to enroll, under prepayment plans, dependents, the self-employed, domestics, farmers, those retired under pension plans, the unemployed, and migratory workers.
4. That hospitals make greater efforts to obtain additional support from individual communities, corporations, and foundations. And that (if governmental help is necessary), federal assistance should be sought only when the state is unable to help.
5. That medical schools and hospitals should accept responsibility for educating physicians and other responsible personnel in hospital economics in an effort to eliminate waste of facilities and supplies.

In connection with the last recommendation, Miss Carolyn K. Winters, administrator of the New England Hospital, believes that everyone connected with a hospital should also be educated in good public relations. This need was emphasized in an editorial that appeared in the *Boston Herald* ("Humanizing the Hospitals," January 11, 1955):

> The average hospital today needs a lot of humanizing. . . . It seems both fair and wise that we who support the hospitals should share also in their day-by-day existence, that we should feel a sense of common purpose with them. We can't if they are aloof.

The 1949 interim report of the New York Hospital Study, directed by Dr. Eli Ginzberg, of Columbia University, predicted that hospitals will face additional financial difficulties: "Hard times, if they come, may force the opening of hospitals to all doctors of a community on an equal basis in order to fill empty beds." The report further emphasized the fact that the best possible service cannot be provided for the public if doctors without staff appointments are refused beds on the grounds that only certain doctors are entitled to a preference in the hospitalization of their patients. In any long-range planning, the report continues, the liberalization of the present, closed "gentlemen's club" system of staff membership should play an important part.

The Massachusetts Medical Society has made a forthright and enlightened effort to do just what the New York Hospital Study recommends by sponsoring what has been called the Gallupe Plan, under which graduates of unapproved medical schools who have no staff privileges in approved hospitals are given an opportunity to practice in hospitals under the supervision of staff physicians. When they are found to be qualified, staff appointments should follow. Where this plan has been put into effect it has worked satisfactorily. A general acceptance of the Gallupe Plan would increase the proficiency of the physicians affected and gain their good will and that of their patients.

Conclusions

My own opinions concerning the voluntary hospitals (which have been liberally enriched by others!) should by now be fairly apparent. Even so, it might be well for clarity's sake, briefly to review them here.

The public, which has already demonstrated its willingness to support the voluntary hospital, should be further acquainted with hospital problems and the need for greater financial assistance.

Every effort should be made by the profession to encourage the expansion of prepayment health insurance, including employer participation.

Staff physicians should concern themselves with the administrative and financial problems of hospitals. "Medical economic auditing" by the staff, for example, may disclose methods by which economies would be possible.

Staff representation on governing boards would make for closer co-operation between staff and trustees.

The cost of hospitalization to the patient should be kept as low as is consistent with the best possible care. This can be accomplished by financing research and teaching from sources other than the patient; integration of hospitals on a regional basis; and utilization of hospital facilities on a basis of six or seven days a week.

The standard of medical practice would be raised and public relations improved by liberalizing staff appointments. The public is entitled to free choice of hospitals as well as of physicians. As American Medical Association President Walter B. Martin has said so well, "Medicine belongs to the people. We are merely its purveyors."

Since the voluntary hospital is an indispensable medium by which the medical profession renders service to the public, its problems must continue to receive the persevering support of both the public and the profession. As one of my favorite sources, Benjamin Franklin, has observed: "Diligence is the Mother of Good Luck. And God gives all things to Industry."

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THE SHATTUCK LECTURE

THE PORTAL CIRCULATION*

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BOSTON

AS part of the pleasant task of preparing this, the sixty-fifth George Cheyne Shattuck Lecture, I reviewed the provisions of Dr. Shattuck's bequest to this society. These state that this lecture should be given "by some suitable person . . . on the climate of the Commonwealth, on the diseases of its inhabitants, and on such other subjects as the said society may select." Needless to say, I have entertained a number of misgivings about the suitability of the person selected for the honor this year, for as I review the accomplishments of my illustrious predecessors, mine seem meager indeed. Because my short acquaintance with the climate of the Commonwealth hardly entitled me to discuss this subject, I was pleased and relieved when the Society graciously accepted my topic, "The Portal Circulation." A year or so ago I might have doubted that this would be a suitable one, for I had had little opportunity to learn whether the ills of this particular vascular system require as much attention among the inhabitants of Massachusetts as among those of New York City. Any doubts I may have had on this score have been dispelled during the two years that it has been my privilege to live in this state.

The portal circulation of living man, lying as it does deep within the abdominal cavity and without readily available external communications, has long defied direct investigation. The very inaccessibility of this unique venous system, which lacks valves and begins and ends in a capillary bed, has contributed immeasurably to the quality of mystery that the complexities of its normal physiology have acquired. Consequently, the many ills to which it and its intimately related organs are subject have, for many years, been poorly understood indeed. During the past ten or fifteen years, however, medical, surgical and research technics have been developed that have brought the portal vein in health and disease under closer scrutiny than heretofore possible. A start at least has been made toward solving a number of the more obvious problems arising in connection with this vascular system.

HISTORICAL BACKGROUND

Historically, it would be misleading to suggest that the portal vein was not well known to our predecessors in medicine. Their observations, of necessity, however, were made either in animals or in man at the autopsy table. Primitive medicine men and the sophisticated soothsayers of ancient civilizations recognized this vessel as originating in the liver. These men held the portal vein in high regard and attached particular significance to the manner in which blood flowed from it at the time of animal and, I suspect on occasion, even human sacrifice. Galen, who considered the liver the source of all animal heat and sanguinification, believed the portal vein to be an important distributing vessel. Vesalius and Fabricus both described the portal vein accurately, but such men as these wrote in ignorance of the true significance of what they observed, for it was not until 1628, when Harvey described the motion of the blood, that, for the first time, important insight into portal blood flow was achieved.

Only twenty-five years after Harvey's epochal discovery Glisson, at a dissection in London, inserted a goose quill into the portal vein and, by means of a pig's bladder, injected thin milk liverward. The liver paled unmistakably as the milk traversed it on its way to the vena cava, to the heart and finally to the lungs. With this dramatic demonstration of the vascular nature of the liver, the portal vein was clearly established as that vessel by way of which blood was collected from the spleen, the pancreas and the gastrointestinal tract and returned thereby to the systemic circulation.

During the late seventeenth and early eighteenth centuries, the portal circulation was the object of much serious study and speculation. In connection with these early investigations, Diemerbroeck's[1] comments, recorded in 1672, are not only amusing but instructive as well, for they provide insight into the fact that at long last medical thinking appeared to be free of Galen's dicta. Of the portal circulation, Diemerbroeck wrote:

> The Vena Porta enters the Hollow Part of the Liver between the two Eminences, which Hippocrates calls πύλας, or Gates. . . . The beginning of this Vein, is by some derived from the Liver, by others from the Mesentery. But the Doubt is easily resolved, by saying that it takes its Rise from the Intestines and the Mesentery both. . . . The Vena Porta by the Physicians is assigned to several Uses. For the Ancients asserted that through their Veins and the Mesaraics [mesenterics] the blood flowed for the Nourishment of the Intestines and other parts contained in the Abdomen; that the Chylus also ascends through the same passages to the Liver. . . . But Dr. Harvey's discovery of the Circulation of the Blood has scatter'd all these Mists of Error; So that now adays there is no man vers'd in Dissection but will deride these Vanities. . . . As to the Motion of the Chylus and the Blood moving upward and downward through the Mesaraics [mesenterics] 'tis contrary to sense; since such a contrary Motion of two different humors can never be at the same time in those Vessels so extreamely narrow. Nor will the similitude

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signifie anything of shavings of Iron and Straw mix'd together in one Pipe, and putting a Load-stone at one end to draw the Iron, and a piece of Amber at the other to draw the Straw. . . . Nor are there any two such different Magnets belonging to the Mesaraics [mesenterics], to draw the Chylus upward and the Blood downward. . . . Others affirm the Blood and Chylus to pass by turns . . . which is ridiculous. . . . Now then the true use of the Vena Porta is threefold.

1. To receive the Blood of the Birth. . . .
2. To conveigh to the Liver and the Hollow Vein the Blood which is forced to the intestines. . . .
3. To conveigh to the same place, the Arterious Blood. . . .

Gradually, centuries of speculation gave way to scientific observation and experimentation. For instance, in 1793, Mr. Abernethy,[2] of London, reported, in the *Philosophical Transactions* of that year, the autopsies of 2 young women who had died of causes unrelated to their portal circulations. In each, however, the portal vein entered the vena cava directly rather than the liver. Aside from being 2 of the first well documented congenital anomalies of the portal vein, these autopsies apparently caused a sensation in medical circles, for they enabled Mr. Abernethy[3] to end, once and for all, an important controversy of the day that centered about whether both portal venous and hepatic arterial blood were necessary for the formation of bile. Because the biliary tracts of both these young women were normal, it was obvious to all that bile could be fabricated from hepatic arterial blood alone.

For over a hundred years, little important reference to the hemodynamics of the portal circulation of man can be found; animal experimentation, however, flourished. Perhaps the earliest significant studies on the portal circulation of rabbits, cats and dogs are those of such eminent physiologists of the last century as Oré,[4] Schiff[5] and Claude Bernard.[6] The genesis of Oré's original experiments is interesting, for these derived directly from the observation that when patients died of cirrhosis of the liver the portal vein was often occluded by thrombus. It was Oré's thought that this disease might be due to deprivation of the liver of its normal portal blood flow. Accordingly, he set out to produce cirrhosis in animals by occluding their portal veins. From Oré's investigations, as well as from those of his contemporaries, it was quickly learned that common laboratory animals died promptly if their portal veins were suddenly and completely occluded. Although Claude Bernard suspected that death under these circumstances might be due to pooling of blood in the splanchnic bed, most observers believed that the cause of death was obvious, hepatic failure. The medical literature of the day is replete with articles likening the death of these animals to poisoning by an overdose of some such lethal drug as morphine. One of these articles, incidentally one by Lautenbach[7] in the *Philadelphia Medical Times* of 1877, was abstracted in a Russian army medical journal later in the same year and came to the attention of Nicolai Eck.[8] This sturdy medical individualist was highly skeptical of the convictions of the day and set about proving that the dog, at least, could survive sudden and complete portal occlusion provided portal blood flow was simultaneously diverted into the vena cava through his now world-famous fistula. Eck proved his point and went off to the army, never to be heard from again, at least so far as the scientific world has been able to determine.

In addition to proving that his dogs could survive, Eck believed that his animals were quite normal. Pavlov,[9] however, became deeply interested in the Eck-fistula dog, and observed that a number of the animals in his experimental colony manifested a serious metabolic disorder to which not a few succumbed. Since death was particularly prone to occur after the dogs had eaten heavily of meat, a syndrome was recognized that has come to be known as "meat intoxication." That Eck also had man in mind when he devised his fistula is evident from his original article: "I am conducting these experiments with the purpose of clarifying some physiological problems as well as to determine whether it would be possible to treat some cases of mechanical ascites by means of forming such a fistula."

From these early experiments, all performed before 1900, two important clinical convictions were derived. In the first place it was firmly believed that man, even as the dog, would die immediately if his portal vein was suddenly and completely occluded. Secondly, it was widely held that the abdominal ascites associated with cirrhosis arose directly from the congestion present in the splanchnic bed. Not, however, until many years later was it appreciated that important elevations in portal pressure often accompany cirrhosis of the liver, as well as primary thrombosis of the portal vein. It is these three aspects of the portal circulation to which I wish to devote the remainder of this discussion — namely, sudden and complete occlusion of the portal vein in man, the site of ascitic-fluid formation in patients with cirrhosis, and portal hypertension.

Occlusion of the Portal Vein

My initial interest in portal occlusion in man appeared during my efforts to improve the rate of resectability in cancers arising in and about the head of the pancreas.[10] In a discouragingly large number of patients harboring primary pancreatic neoplasms, I found the portal vein or the superior mesenteric vein or both invaded by tumor (Fig. 1*A* and *B*). It occurred to me that if these vessels could be included when pancreaticoduodenectomy was performed for primary pancreatic cancer, some measure of improvement might be added to this operation, the accomplishments of which had been none too brilliant. Two methods of achieving this objective seemed reasonable: first, resection of the portal vein and simple abrupt ligature of the superior mesenteric venous radicles; and resection of the portal vein and con-

comitant diversion of portal flow into a neighboring vessel such as the vena cava.

Shortly before I became interested in this problem, Whipple and his associates[11,12] had demonstrated that in patients with cirrhosis at least the entire portal flow could be successfully diverted into the vena cava. I could not be sure, however, that patients whose livers were normal could be expected to tolerate such my first experimental animal, I was forced to ligate this vessel suddenly and completely.[13,14] Such was my heritage in surgery that I sadly returned this animal to its cage convinced that he would die shortly. Much to my surprise and that of my associates, this monkey survived apparently undisturbed by the operation. We then became interested in why the monkey survived sudden and complete portal occlusion

A B

FIGURE 1. *Portal Venograms.*

A = normal roentgenogram obtained immediately after injection of 40 cc. of 70 per cent Diodrast into the superior mesenteric vein. This vessel, together with the portal and the intrahepatic portal radicles, is clearly outlined. (Portal pressure is equivalent to 14 cm. of saline solution.)

B = abnormal roentgenogram demonstrating obstruction of the portal vein by a cancer arising in the head of the pancreas. The extensive collateral vessels bypassing the point of obstruction are clearly visualized. (Portal pressure is equivalent to 23 cm. of saline solution.)

sudden deprivation of portal blood. I believed, therefore, that before experiments on man should be considered the several problems involved in resecting the portal vein en bloc with the head of the pancreas should be studied in the research laboratory. The *Macaca mulatta* monkey was chosen as the experimental animal, for the anatomic relations about the head of the pancreas, the duodenum and the portal vein in this primate are identical with those of man. It was my original plan to fashion in one stage an end-to-side Eck fistula and then to resect the head of the pancreas and duodenum together with the portal vein. Because I irreparably tore the portal vein of whereas the dog did not. By portal venography and manometry, we demonstrated that portosystemic collateral vessels were immediately available to the monkey through which portal blood escaped into the general circulation in sufficient quantity to support an effective circulating blood volume. The dog, we found, is not endowed with such collateral vessels and dies promptly, therefore, by bleeding to death into the splanchnic venous bed. So extensive is this collateral circulation in the monkey that the initial portal hypertension produced by portal occlusion subsides within a week to ten days.

After demonstrating on innumerable occasions that

the monkey survived sudden and complete portal occlusion, we cautiously applied the same experiment to patients who were discovered at exploratory celiotomy to harbor inoperable cancers of one variety or another. Such persons also survived this experimental procedure.[14,15] So far as his portal circulation at least was concerned man was found to behave much more like a monkey than a dog. Figures 2*A* and *B* reproduce two portal venograms, one in man and one in the monkey, illustrating a few of the portosystemic collateral channels available immediately to these two species for autodecompression of their occluded portal circulations into their systemic venous beds.

A B

FIGURE 2. *Portal Venograms.*

A=*film taken after sudden and complete occlusion of the portal vein by ligature in the* Macaca mulatta *monkey. (Portal pressure equivalent to 49 cm. of saline solution.)*

B=*film taken after sudden and complete occlusion of the portal vein by ligature in man. (Portal pressure equivalent to 39 cm. of saline solution.)*

The application of these experimental data to patients with operable cancer of the pancreas invading the portal vein is a technical story,[13-16] the details of which have been published and need not be discussed here. Suffice it to state that resection of the portal vein in continuity with the head of the pancreas is now a feasible surgical operation that has been made a matter of record not only by me but also by McDermott and Adams,[17] Moore[18] and others. In making this statement, I must not, however, be guilty of suggesting that inclusion of the portal and superior mesenteric veins in pancreaticoduodenectomy has materially improved the lot of the patient with pancreatic cancer. In my experience it has not, and the operation appears to be justified only in the rare patient whose portal vein is invaded by tumor and who manifests neither regional nor distant metastases. Regarded, however, as a study in the physiology of the portal circulation in man, this operation has proved that certain persons whose livers are normal can survive sudden and complete occlusion of the portal vein. It has also provided insight into how effectively the portosystemic collateral vessels with which man is naturally endowed can decompress his own portal bed. Furthermore, it has served to explain some of the many difficulties encountered in attempts to produce portal hypertension experimentally. Apparently, the portal circulation of primates, transiently rendered hypertensive by portal occlusion, manifests an almost infinite capacity for returning to normotensive levels. Appreciation of

this fact, of course, has been one of the important stumbling blocks in the way of explaining satisfactorily why sustained elevations in portal pressure are encountered in patients whose portal blood flow meets obstruction either within or without the liver.

Site of Ascites in Cirrhosis

On these observations on the physiology of the portal circulation in the monkey and in man can be based certain thoughts bearing directly on my second topic, the site of origin of abdominal ascites consequent to cirrhosis of the liver. As mentioned above, Eck, as well as many others, believed that this type of ascites was due to splanchnic congestion or, to use a more modern and exact term, portal hypertension. Evidence that ascites is not due to portal congestion or hypertension can be derived from various and well documented sources. For instance, in patients with normal livers but with portal hypertension and esophageal varices due to primary portal venous thrombosis, abdominal ascites is rarely if ever encountered. Figure 3 reproduces the portal venogram of a thin young woman suffering from variceal hemorrhage due to extrahepatic portal thrombosis. Although the portal pressure was equivalent to 41 cm. of saline solution there was never any evidence of abdominal ascites. Furthermore, it is well recognized that many patients with cirrhosis, esophageal varices and verified portal hypertension never manifest ascites. Also, in any number of patients with widely patent portacaval shunts, a circumstance that precludes the development of portal hypertension, ascites has been observed to develop as a direct manifestation of hepatic decompensation. Finally, in this connection, I should like to emphasize the fact that ascites has not appeared in any of the monkeys or in any of the patients subjected to sudden and complete occlusion of the portal vein, even though all these subjects manifested severe, though transient, elevations in portal pressure.

So much for negative evidence. What is available to indicate positively where ascites in cirrhosis does come from, since it apparently is not derived from the congested and hypertensive splanchnic venous bed? I believe that some recently reported experiments and observations are crucial in this regard. Both Freeman[19] and Mallet-Guy et al.[20] have transplanted the livers of dogs into the pleural cavities of the animals. After partial occlusion of the hepatic venous outflow tracts, a technic well known to produce portal hypertension and massive abdominal ascites, intrathoracic collections of ascitic fluid developed. Although these experiments have not yet been widely confirmed, I consider them sufficiently critical to warrant the conclusion that the primary source, at least, of this type of experimental ascites is from the surface of the liver itself. Hyatt and Smith,[21] sharing similar beliefs, placed a plastic bag beneath the lobes of the exposed liver and partially occluded the vena cava just below the diaphragm. Ascitic fluid oozed from the surface of the liver and dripped into the collecting bag. These laboratory observations are supported by the recent morphologic demonstration of Madden et al.[22] that where the cirrhotic obstruction

Figure 3. *Portal Venogram in a Young Woman with a Normal Liver and a Level of Portal Hypertension Equivalent to 41 Cm. of Saline Solution Owing to Thrombosis of the Portal Vein.*

This vessel was replaced by a racemose collection of thin-walled veins corresponding to the older description of "cavernomatous transformation" of the portal vein. In spite of the elevated splanchnic venous pressure, the abdominal cavity was free of ascites.

to portal flow resides predominantly in the hepatic outflow tracts, massive ascites is prominent. Furthermore, it is well known that most troublesome ascites is associated with Chiari's syndrome. Here, of course, the primary pathologic process is obstruction of the hepatic veins. Today, then, there is evidence that ascitic fluid is extravasated from the surface of the liver. It is not at all improper, therefore, to state that ascites in cirrhosis is due to *intrahepatic venous hypertension* rather than to *splanchnic venous hypertension.*

At this point I should be remiss indeed if I did not acknowledge that the most important factors predisposing to ascitic-fluid formation in patients with cirrhosis are lowered osmotic pressure due to depleted serum proteins, particularly albumin, abnormalities in metabolism of salt and water and more than likely a disturbed hormonal balance. From an analysis of their data in fifteen carefully controlled metabolic

studies of patients with cirrhosis and ascites, Habif, Randall and Soroff[23] were forced to the conclusion that "While portal hypertension is probably a factor in the formation of ascites, its role must be minor, as witness the majority of patients with cirrhosis and portal hypertension without ascites." Furthermore, these investigators, as well as Eisenmenger,[24] Patek and his associates[25] and many others, have demonstrated that a diet rigidly low in sodium, moderate in protein and high in calories, in the majority of cases, controls ascites without at the same time providing any reason to believe that portal hypertension has receded. In fact, quite the opposite, for, as Habif has pointed out, "the mean reading in the group without ascites was 400±90 mm. of water, and in those with ascites (minimal to severe) it was 370± 30 mm. of water."[23] These metabolic and physical facts, however, relate to the circumstances under which ascites makes its appearance, not to its site of origin.

Here it may well be asked whether under any circumstances portal decompression should be considered for the treatment of ascites. If asked this question, I should have to admit that I should still accept for operation the rare patient in whom persistent ascites develops in the face of good general health, who manifests ability to handle salt well and, most particularly, who is able to maintain a normal level of serum albumin. Perhaps I still cling tenaciously to the old belief that somehow or other splanchnic congestion plays some part, however small, in ascites formation. Perhaps, on the other hand, I happily believe that by reducing pressure on the portal side of the hepatic sinusoid, I simultaneously decrease the degree of hepatic venous obstruction, promoting thereby hepatic venous outflow.

Portal Hypertension and Bleeding Varices

Portal hypertension and bleeding esophageal varices in patients with cirrhosis of the liver constitute the third problem that it is my privilege to discuss this morning. Today, this subject is one of wide clinical interest, and one in which the pathologic physiology of the portal circulation figures importantly. Credit for insisting that cirrhosis is associated with portal hypertension, and that the Eck fistula should be effective in treating patients with varices, properly belongs to McIndoe.[26] In 1928 this investigator wrote as follows:

> Considering that the whole tendency of portal cirrhosis is to divert the stream of portal blood away from the liver, . . . the most logical and effective method of dealing radically with the embarrassed portal circulation would doubtless be to perform a simple Eck fistula. By this means the portal hypertension would be relieved, stasis immediately abolished, and the development of varices arrested.

Within twenty years Whipple,[11] Rousselot,[27-29] Blakemore and Lord,[12] Linton[30] and Linton and his associates[31] proved that portal hypertension was a verifiable entity in living man, and that elevated levels of portal pressure could be lowered significantly by means of a shunt between the portal and systemic venous circulations. In 1945 Whipple[11] wrote as follows of his initial efforts to decompress the hypertensive portal system:

> These procedures are as yet purely experimental. They have been carried out in patients that had had repeated severe hemorrhages, and for whom conservative measures offered no hope. . . . It will require a follow-up period of three years, or more, to determine the value of these portacaval short-circuiting procedures. At least they represent a bold attempt to deal with the problem of portal hypertension in its life-threatening forms.

That Dr. Whipple's suggestions were accorded serious attention is reflected in Linton's[30] report of 1951, in which he wrote on the selection of patients for portal decompression as follows: "A portacaval shunt is the only method which reduces satisfactorily the portal hypertension by shunting the portal blood directly into the systemic venous system, thereby reducing the pressure in the esophageal varices and the danger from hemorrhage from them."

A year later, Blakemore,[32] in recording his experiences with 166 shunt operations of this type, made the following comment:

> Of all the surgical procedures that have been employed in the treatment of portal hypertension, the portacaval shunt operation is the one procedure in which the effects upon portal hypertension have been quantitated on the spot by direct portal pressure measurements . . . the follow-up correlation of the subsidence of the esophageal varices with the cessation of hemorrhages postoperatively establishes the rationale of portacaval shunting for portal hypertension.

During the past five years, an appreciable number of reports have been published on portal decompression for variceal hemorrhage secondary to portal hypertension. There is available today, therefore, a considerable experience with this operation. From this a number of significant conclusions may be drawn concerning the effectiveness of this operation in preventing further hemorrhage. It is well known, of course, that varices develop in response to abnormal elevations in portal pressure and that portal hypertension is most commonly due to either portal venous thrombosis or one or another variety of cirrhosis of the liver. Because of marked differences in the type of patient in whom each of these two syndromes is encountered, it is wise, I believe, to consider their treatment separately.

Portal hypertension and bleeding varices due to portal venous thrombosis are primarily a disease of children and young adults whose liver function is, for practical purposes, normal. For this reason, these young patients are excellent surgical risks, and few today question the advisability of portal decompression by whatever means possible when massive hemorrhage becomes a threat to their lives. Unfortunately, successful shunts are difficult to perform in patients with portal thrombosis, and postoperative failure of the anastomosis to function is relatively common.

Nevertheless, a vigorous surgical approach to the control of hemorrhage is warranted in these patients, for their expectancy of prolonged life is excellent provided they can be rid of their bleeding varices. In fact, when all efforts at satisfactory decompression have failed to control hemorrhage, esophagogastrectomy to remove the local site of hemorrhage is often justified, provided the jejunum rather than the stomach is used to re-establish continuity in the gastroenteric canal. It is not in this group of patients whose varices bleed that major controversies over the propriety of portacaval shunts have arisen.

The major disputes over portal decompression concern middle-aged to elderly patients with cirrhosis of the liver who have varices from which massive bleeding takes place. Here is a group of patients who differ importantly from those whose obstruction to portal flow resides outside the liver. Life expectancy is sharply restricted by hepatic disease; addiction to alcohol and a poor diet are common, and because of low hepatic reserve operations of the magnitude of portal decompression may be poorly tolerated indeed. Repeated hematemeses may, on the other hand, be withstood even less well than surgical intervention. Although it may not be possible at present to resolve all the controversies that have arisen over whether a

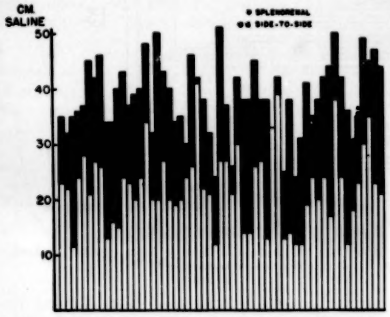

FIGURE 4. *Immediate Fall in Portal Pressure after Opening of an End-to-Side Portacaval Shunt.*

This composite graph clearly demonstrates the fall in pressure obtained after a successful shunt of large caliber. In this series of 56 consecutive patients the average fall was equivalent to 20 cm. of saline solution.

portacaval shunt should be advised for the patient with cirrhosis who bleeds from his varices, there are evidences available today that this operation has at least emerged from its experimental phase. A number of important observations on the physiologic effects of this operation can therefore be made, and to these I wish to devote the remainder of this presentation.

In the first place, it can now be said without serious fear of contradiction that a shunt of large caliber successfully established between the portal and caval vascular systems reduces an elevated portal pressure to normal or nearly normal levels. For instance, in a consecutive series of 56 patients operated upon at the New York and New England Center hospitals, abnormally high portal pressures were reduced an average of 20 cm. of saline solution by means of an end-to-side portacaval shunt. The degree of fall obtained in each patient immediately after the shunt

FIGURE 5. *Percutaneous Splenoportogram Obtained Some Months after Portal Decompression by Means of an End-to-Side Portacaval Shunt.*

Diodrast, injected directly into the spleen, traverses the splenic vein, passes through the shunt and is rapidly dispersed in the vena cava.

has been opened is graphically portrayed in Figure 4. In the early days of portal decompression much speculation centered about whether such shunts, fashioned as they are between two vessels of relatively low blood pressure, remained patent. By now a sufficient number of patients with major portal decompressions have come to autopsy with their shunts widely open to convince even the most skeptical that these anastomoses do indeed remain patent. Figure 5 reproduces a percutaneous splenoportogram, representative of a number obtained long periods after operation. Here the radiopaque substance can be readily traced from the spleen through the splenic vein into the vena cava. That a successful shunt protects a patient who has bled massively, even on several occasions, from further hemorrhage is also evident from a study of available reports. My own experience, together with that of my immediate associates, in portal decompression now totals 56 patients with cirrhosis and portal hypertension who have been operated on for control of massive variceal hemorrhage; 48 have survived operation for a month to four years. During this period not

a single hemorrhage has occurred* (Fig. 6). Blakemore[22] has described 78 patients with cirrhosis, only 7 of whom have bled since operation. Linton[30] lists only 3 patients out of 27 who have bled since portal decompression. These experiences are not unique and, in my opinion, warrant the conclusion that in patients with cirrhosis a successful portacaval shunt lowers portal pressure significantly, remains patent indefinitely and protects the vast majority of patients who have bled against further hemorrhage.

The exact mechanism by which surcease from variceal hemorrhage is effected by portal decompression must remain a matter of conjecture, however, until

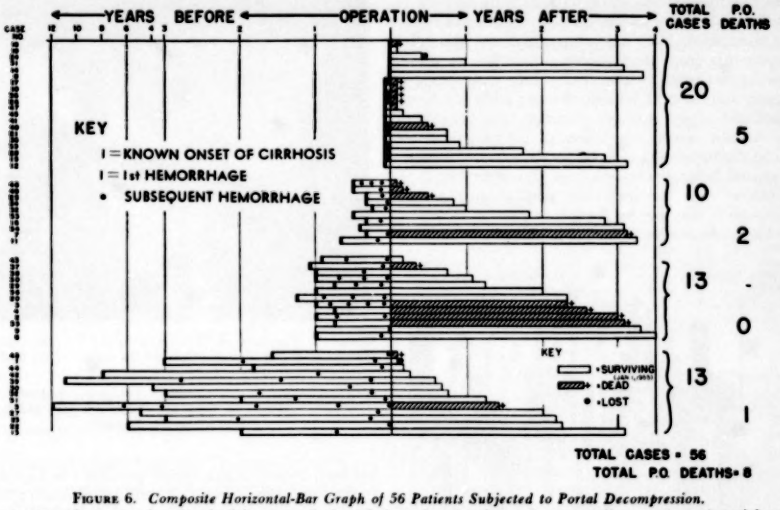

FIGURE 6. *Composite Horizontal-Bar Graph of 56 Patients Subjected to Portal Decompression. The preoperative course is epitomized in terms of elapsed time after first hemorrhage as well as total number of hemorrhages sustained by this group. Postoperative survival is also outlined.*

the precise cause of the hemorrhage has been determined. Certainly, it seems unlikely that mere reduction in pressure within the varix itself is significant in this regard, for it is difficult to believe that varices rupture simply because of increased pressure. Perhaps the most logical explanation available today is that, as a result of lowering of portal pressure, the turgid and tense varices of the lower esophagus and upper stomach subside. Function at the esophagogastric junction is consequently improved, and acid-peptic regurgitation, ulceration and hemorrhage prevented.

*Some idea of the probability of further hemorrhage during the survival period can be gained from the following crude calculation: this group as a whole survived their first hemorrhage for a total period of eighty years before operation. During this time they sustained 120 massive hemorrhages, or an average of 1½ hemorrhages per patient per year. Not 1 patient has bled since operation, although hemorrhages would have been anticipated during the total survival period of eighty years in those who did not succumb to the operation itself.

However, serious as massive hemorrhage is believed to be for the patient with cirrhosis, the prevention of further bleeding is but one aspect of the problems in therapy presented by this unfortunate group of patients. Still to be considered seriously is the question of whether portal decompression improves significantly their period of survival. It seems unlikely indeed that a major operation practiced simply for the control of one manifestation of a disease will long remain popular if the survival period is but a few months and the surgical mortality unreasonable. With doubts such as these in mind, a number of thoughtful physicians and surgeons have recently questioned the propriety of portal decompression. The following quotations representative of such misgivings have been taken at random from more or less recent articles on the subject.

> . . . We have as yet insufficient controlled data to be sure that over-all survival is appreciably different in the group operated upon as compared to the group not undergoing operation. Until we are convinced that operation definitely decreases mortality, we feel required to allow every opportunity for the spontaneous disappearance of varices before recommending surgery.[28]

> Close scrutiny of the results reported in the literature make it reasonable to question whether the operations advocated are really responsible for the good results obtained.[31]

> Bleeding from esophageal varices raises the whole question of the surgery of portal hypertension. I know of no branch of surgery more urgently in need of debunking. The subsequent history of patients who have undergone these operations is, on the average, no better than that of

those who have been untreated and, when they die, the new opening is usually found to have closed.[38]

Portacaval shunts appear to be of doubtful value and produce at best a temporary benefit. There is evidence to indicate that even partial deviation of the portal flow may impair liver function. Further experience is necessary before proper evaluation of this operation can be made.[38]

Such opinions as these clearly indicate that a number of controversial issues have arisen in connection with portal decompression. These, of course, require resolution before the usefulness of this operation can be finally evaluated.

Before examining these conflicts further, I believe I should digress to comment on certain inferences that appear to have been drawn from a comparison of two apparently similar but really quite dissimilar groups of patients. Both of these, of course, have had cirrhosis and variceal hemorrhage in common. In 1942 Ratnoff and Patek[37] and in 1948 Patek and his associates[25] presented their classic studies on the natural history of Laënnec's cirrhosis. From both these studies it was concluded that 50 to 70 per cent of patients with cirrhosis who bleed will be dead within a year to eighteen months of the first hemorrhage. For convenient reference, Ratnoff and Patek's[37] original twelve-month and five-year survival curves after first hematemesis have been reproduced in Figure 7. Others, notably Shull,[38] Snell[39] and Nachlas et al.,[40] have studied similar groups of patients and come to like conclusions. The twelve-month survival curves

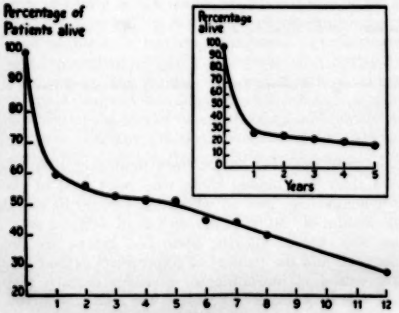

FIGURE 7. *Original (1942) Survival Curves after First Hematemesis (Based on Ratnoff and Patek[37]).*

of Ratnoff and Patek[37] and Nachlas and his associates[40] have been redrawn (Fig. 8, Curves B and C). Summed up together, these studies have provided an opinion, widely held today, that between 50 and 80 per cent of patients with cirrhosis who bleed will be dead within a year. This belief has been popular among those interested in portal decompression. It seems to have provided justification of and stimulation to continued surgical treatment of patients with esophagogastric hemorrhage due to cirrhosis, apparently in an effort to improve this presumed high natural mortality rate. Although I realize that I may do injustice by quoting out of context, I read from two representative articles appearing in recent surgical journals:

90 of these had cirrhosis of the liver and 41 or 45 per cent of them succumbed to hemorrhage. . . . These figures

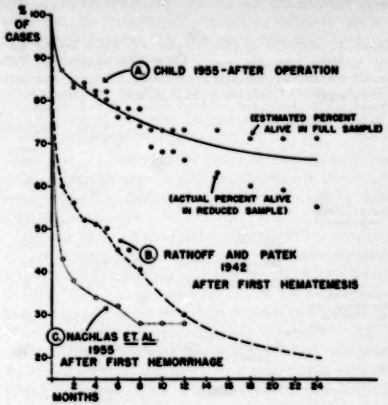

FIGURE 8. *Various Survival Curves.*
A=*survival curve of series at the New York and New England Center hospitals of 56 patients with cirrhosis subjected to portal decompression for variceal hemorrhage.*
B=*(1942) twelve-month survival curve (redrawn) after first hematemesis, according to Ratnoff and Patek[37] (1942).*
C=*twelve-month survival curve after first hematemesis, according to Nachlas et al.[40] (1955).*

demonstrate more clearly than words can express the terrific mortality rate in this group of patients. . . .[39]

For example, Patek in a follow-up study of 150 carefully treated cases of cirrhosis states that 50 percent of the patients who had hematemesis were dead within the 12-month period dating from the onset of their first hemorrhage. Snell reports an even higher mortality; namely 80 percent within a year of their first hemorrhage.[25]

Until recently, I held views not unlike these, and it discomforts me to recall that I have frequently employed similar reasoning to persuade myself and others that patients with cirrhosis who bleed from their varices should undergo portal decompression. Circumstantially, perhaps, but nonetheless prominently, two important connotations of portal decompression have apparently thrived on such random remarks as these: if hemorrhage were not controlled, mortality within the year would be from 50 to 80 per cent, and if further hemorrhage is successfully prevented, survival is prolonged significantly. If ascites, jaundice and impaired liver function are accepted as indications of the degree of severity of cirrhosis, it is obvious

from pertinent data available that these ominous signs of liver disease were far more prevalent among those patients on whom the natural history of cirrhosis has been based than among those who have been selected for portal decompression. For clarity, these comparisons are presented in Table 1. To infer, by comparison of these two dissimilar groups of patients, that 50 to 80 per cent of those treated surgically would have been dead within a year, if a portacaval shunt had not been performed, has in my opinion been unrealistic. role of a "coup de grace." Similarly, if ability to survive operation is accepted as an indication of good hepatic reserve, it is interesting to compare Blakemore's[32] "good-risk" patients, whose operative mortality was 9.3 per cent, with his "poor-risk" patients, 39.9 per cent of whom died of the operation. In other words the more advanced the liver disease the less well do patients tolerate either hemorrhage or operation. From these analyses it has appeared logical to me to conclude that cirrhosis among those who

TABLE 1. *Severity of Cirrhosis on Admission among Six Groups of Patients (Three Treated by Portal Decompression and Three Representing the Natural Life History of the Disease).**

GROUP	AUTHOR	YEAR	ASCITES	SERUM BILIRUBIN		SERUM ALBUMIN		CEPHALIN FLOCCULATION		BROMSULFALEIN RETENTION	
				VALUE OBSERVED	FREQUENCY OF OCCURRENCE	VALUE OBSERVED	FREQUENCY OF OCCURRENCE	VALUE OBSERVED	FREQUENCY OF OCCURRENCE	VALUE OBSERVED	FREQUENCY OF OCCURRENCE
			%	*mg./100 cc.*	%	*gm./100 cc.*	%		%	%	%
Surgical (portacaval shunt):											
	Linton[30]	1951	32.0	>1.0	60	<3.0	12.0	+ + + to + + + +	72	—	—
	Blakemore[32]	1952	41.5†	>1.5	25	<3.0	13.0	—	—	>35	30
	Child et al.[34]	1955	34.0	>1.5	43	<3.0	7.2	+ + + to + + + +	40	—	—
Nonsurgical (natural life history of cirrhosis):											
	Ratnoff & Patek[27]	1942	78.0	(Clinically jaundiced)	65	—	—	—	—	—	—
	Patek et al.[25]	1948	93.0	(Clinically jaundiced)	67	<3.5	90.0‡	Positive	94	>10	100
	Nachlas et al.[40]	1955	62.0	(Clinically jaundiced)	40	Decreased	31.0	+ + + to + + + +	61	>35	55

*In spite of certain vagaries due to variations in methods of reporting figures considered to provide reliable estimate of differences in severity of cirrhosis among 6 groups concerned.
†See Habif's figures on ascites as reported in Habif et al.[33]
‡Average on admission of all patients, 2.8 gm. per 100 cc.

Additional differences between the two groups of patients become apparent when a few further facts concerning them are considered. For instance, fully 50 per cent of the patients operated on at the New York and New England Center hospitals had demonstrated preoperatively an ability to survive first hemorrhage by six months or longer (Fig. 6). Furthermore, as a group, these patients were selected for operation because, in addition to massive hematemesis, they presented at least a reasonable degree of hepatic functional reserve. In this immediate connection it is of interest that in each of the series of patients described by Patek and his associates (1942[27] and 1948[25]) a number of robust persons, presumably with good liver function, survived the first hemorrhage for many months — a few as long as five years (Fig. 7). The authors remarked upon the striking contrast between these hardy patients and those who died after the first hemorrhage.[41] Hemorrhage among the former group appeared to be an accident and was well tolerated; among the latter, massive bleeding played the have been operated on has been significantly less severe than that among those who have died of the first hemorrhage and on whom what might be called the dictum of "50 to 80 per cent dead within a year" has been based. Clearly, these two groups are not identical, and the validity of impressions gathered by comparison of mortalities or survivals in each may justly be questioned. Therefore, the readily available survival curves of the natural history of cirrhosis after first hemorrhage should not, in my opinion, be employed as standards against which to measure the ability of portal decompression to prolong life.

I must now consider whether the demonstrated ability of portal decompression to prevent further hemorrhage has favorably or unfavorably influenced the over-all survival of a group of 56 patients with reasonably well compensated cirrhosis, all of whom have bled on at least one occasion. In this regard my first obligation is to point out as accurately as possible just what has happened to these selected patients after they have been subjected to portal de-

compression for the purpose of preventing further hemorrhage and for the alleged purpose of prolonging their lives. I believe I have shown conclusively that further hemorrhage has been prevented. Now I must examine the supposition that life has thereby been significantly prolonged. Postoperative survival may be conveniently expressed in two ways: by means of a horizontal-bar graph that summarizes the preoperative course of each patient for hemorrhage and the postoperative course for survival (Fig. 6); and, a curve of survival after operation plotted in terms of percentage of the original population alive at the end of arbitrarily chosen periods (Fig. 8*A*). Examination of these two figures promptly reveals an immediate mortality of 14.2 per cent ascribable to the operation itself.* Generally, this occurred within a day or so to a month of operation and was not due to hemorrhage. Thereafter, members of the original population have disappeared as they have succumbed, presumably at the natural attrition rate of their primary disease, but again not of hemorrhage. At the end of a year approximately 72 per cent of the original group have been alive, whereas by the end of two years the survivors have decreased to 66 per cent. Interestingly enough, the general slope of this curve is not unlike that manifested by the several curves adduced to represent the natural history of cirrhosis after first hematemesis (Fig. 7 and 8*A*, *B* and *C*). Again, however, a warning against drawing conclusions from a comparison of these two curves is necessary for, as I believe I have shown above, evidence is insufficient to consider identical the two groups of patients on which each of these curves has been based. Furthermore, first hemorrhage is not synonymous with portal decompression. For practical purposes these two exhibits reflect a surgical mortality of 14.2 per cent and an attrition at the end of two years of an additional 19.8 per cent. For convenience, it may be said that in this series of 56 surgically treated patients 34 per cent were dead at the end of two years. Because an insufficient number of patients in this series have been followed long enough, it is not considered proper to extend their survival curve beyond the two-year period.

Before a final conclusion can be drawn about the influence, favorable or unfavorable, of portal decompression on over-all survival, some suitable standard of measurement must be acquired. Unfortunately, I have been unable to find data on which to base an appropriate survival curve of patients in reasonable hepatic reserve who have bled but who have not been treated by portal decompression. Only when such information as this becomes available will it be possible to fashion a standard against which to measure the success or failure of Dr. Whipple's "bold attempt to deal with the problem of portal hypertension in its life-threatening forms." I am hopeful that in the near future Dr. Linton, of Boston, and Dr. Blakemore, of New York City, may resolve the many controversies that have arisen over the usefulness of portal decompression. Their experience now numbers many patients with this distressing problem, and I eagerly await their present conclusions.

At this point I am reminded of a lively discussion that took place a number of years ago among Drs. Whipple, Orr and Horsley[42] over whether pancreaticojejunostomy should be performed in connection with enteric reconstruction after subtotal pancreaticoduodenectomy. Dr. Horsley spoke as follows:

> The physiologists have shown that the external pancreatic secretion is not essential to life. However, like the old jingle that poverty is no disgrace, it certainly might be claimed that it is sometimes inconvenient. It certainly is convenient to have the external secretion preserved if it can be accomplished.

With massive hemorrhage from varices, as with poverty, it may be no disgrace, but certainly it is inconvenient and a great comfort indeed if it can be legitimately prevented.

If these were all the factors concerned one might resolve the controversial aspects of portal decompression for variceal hemorrhage merely by asking the patient whether he would like to be rid of his hemorrhages, fully realizing, however, that at present it is not possible to predict whether his life expectancy will be significantly prolonged or perhaps even shortened. Such a superficial approach to a problem as serious as this one constitutes, of course, wholly unjustified oversimplification of the many intangibles that unfortunately still exist today. Granted that a given patient with good hepatic reserve might wish to avoid a surgical mortality of 10 to 15 per cent and take his chances on further hemorrhage, what assurance can be given him that he will not bleed again or that, if he does, he will survive his next hemorrhage? Such evidence as is available strongly indicates that he will bleed again. For instance, in the series at the New York Hospital and New England Center Hospital (Fig. 6), 30 patients who survived the first hemorrhage for six months to twelve years bled on an average of one and a half times a year. A few such patients may, of course, not bleed again but, as yet, criteria on which to predict the probability of future hemorrhage in any given patient are too imperfect to permit forecasting which ones will bleed again and which ones will not. Nor can the mortality rate of future hemorrhage, if it occurs, be estimated with any accuracy at present. In this connection it is interesting to recall again that, in 1948, Patek et al.[28] wrote that although approximately half of 42 patients died within a year of the first hemorrhage, there were some who survived hematemesis for a long period. In this group of 22 patients who died 6, according to these authors, were in a good state of hepatic reserve whereas the remaining 16 were in obvious hepatic failure. In the same series of medically treated patients, 9 survived five years or longer without recurrence of hematemesis.

*Seven patients who died within a month and 1 patient who died within two months of operation have been selected to represent the immediate surgical mortality, even though 1 or 2 of these actually left the hospital alive.

On the other hand, and perhaps in favor of a patient's taking his chances with further hemorrhage, is experimental evidence that deprivation of the cirrhotic liver of its portal blood may be an insult that such a liver can ill afford. For instance, it is well known that in the dog an Eck fistula not only deprives the liver of its ability to regenerate but is even associated with hepatic atrophy.[43] G. H. Whipple[44] has shown conclusively that the Eck-fistula dog is under a distinct handicap so far as his ability to fabricate certain essential proteins is concerned. That complete shunting of portal blood away from either the normal or the cirrhotic liver may be followed by a serious defect in ammonia metabolism seems evident from the number of studies on this subject that have recently been reported.[45-47] As when an attempt is made to predict further hemorrhage, it seems impossible to foretell which patients will have symptoms of ammonia intoxication after portal decompression and which ones will not manifest this undesirable sequela of the operation. Nor has it been finally determined whether the faulty ammonia metabolism that may occur after an end-to-side portacaval shunt is due to the shunt itself or to progression of the cirrhosis. Bearing these possibilities in mind, those who relate episodic stupor to the end-to-side portacaval shunt have persuaded at least a few surgeons to revive interest in the more extensive use of the splenorenal shunt, a type of portal decompression that not only effectively lowers portal pressure but also, presumably at least, permits a modicum of portal blood to continue to flow through the liver.

In view of these many controversial aspects of portal decompression, what logical approach may be taken today by those charging themselves with the management of patients with cirrhosis who are bleeding or who have bled massively from their esophagogastric varices? Briefly, I believe that until surgical risk is proved to be significantly greater than the risk of death from further hemorrhage, I shall continue to advise portal decompression for the patient with cirrhosis whose hepatic reserve conforms generally to the criteria for selection of patients for operation that were aptly proposed by Linton[30] a few years ago. In general, jaundice should be minimal or absent, and ascites should not be a feature of the disease. Patients selected for operation should be able to maintain a serum albumin of 3.0 gm. per 100 cc. or higher, and they should not have been in hepatic coma. If one or more signs of hepatic failure appear after recovery from massive hematemesis, the patient should be given time to demonstrate that his liver can recover sufficiently to conform to these well established criteria of operability.

Patients who present themselves for care during an episode of massive hematemesis constitute problems in therapy that may tax medical and surgical judgment and ingenuity. Since neither the diagnosis of cirrhosis per se nor the general management of patients with sudden and severe upper gastrointestinal hemorrhage falls within the scope of this discussion, I shall assume here that a diagnosis of bleeding varices has been made, that hemorrhage has been controlled by pneumatic tamponade and that blood replacement has been adequate for the control of shock. What should be done next in the patient's behalf? First of all, I believe that an effort should be made forthwith to evaluate the patient in terms of hepatic reserve. If hemorrhage has appeared suddenly in a patient who, for months or perhaps even years, has been on the borderline of frank hepatic failure, I believe that one should watch and wait. It is such patients as these who, I am sure, contribute to the high initial mortality of first hematemesis in cirrhosis (Fig. 8*B* and *C*). It is difficult for me to believe that heroic operations of magnitude will ever improve the mortality figures in this group of patients whose hemorrhage conforms to Patek's "coup de grace."

In sharp contrast to these patients whose hemorrhage is all but the terminal episode are those whose bleeding has appeared suddenly in a setting of good hepatic reserve. Perhaps such a person has been working steadily, has never been jaundiced and has never had ascites or other stigmas of hepatic failure. Liver function in such a patient as this can be evaluated on an emergency basis. If the serum bilirubin is 1.5 mg. per 100 cc. or below, if the serum albumin is 3.0 gm. per 100 cc. or above and if the patient is alert and does not have ascites, I believe today that a portacaval shunt should be performed as an emergency procedure with pneumatic tamponade in place. Effective as prolonged dietotherapy and salt restriction have proved to be in improving the general outlook of the patient with cirrhosis, it is inconceivable to me that this form of treatment will ever find a place in the immediate control of massive hemorrhage.

In an effort to improve the survival rate in this group of patients, emergency portal decompression was performed on 8 patients in the series at the New York and New England Center hospitals (Table 2). Three of these patients died in the immediate postoperative period; of the remaining 5, 3 survived for six, thirty-nine and seventeen months respectively before succumbing to hepatic failure, and 2 have survived for nine and twelve months respectively, in spite of the fact that all patients were moderately jaundiced. All but 2, however, had a serum albumin of 3.5 gm. per 100 cc. or above. Two of the three deaths occurred in patients with levels below 3.0 gm. per 100 cc. Although this series is admittedly too small to permit final evaluation of portal decompression as an emergency operation, it does indicate that the operation can be performed successfully and hemorrhage satisfactorily controlled even under emergent circumstances. Although Crile,[48] Linton and Warren[49,50] and others have advocated direct transthoracic-transesophageal ligation for the emergency control of hemorrhage, I have never been particularly enthusi-

astic about this procedure, which has seemed to me to be almost as major an undertaking as a shunt. This view of mine is shared by Johnson,[51,52] who recently made this aspect of portal decompression the subject of an address before a regional meeting of the American College of Surgeons. Furthermore, since direct ligation is not a definitive operation and must be followed by portal decompression at a later date, a patient who is admittedly a poor risk must be subjected to the hazard of two major operations rather than one. Just as elective portal decompression had to be regarded as an experimental procedure when first used, so must a shunt performed on an emergency basis be considered today.

One group of patients remains to be considered — those who are discovered to have varices but who have never bled. Today some enthusiasm is being expressed for portal decompression in this group, provided the patients conform to the generally accepted criteria for elective operation. In advocating surgical intervention under these circumstances, Palmer and his associates[53] have expressed the following belief:

> . . . 20 per cent of cirrhotic patients whose varices were demonstrated without history of previous hemorrhage bled annually thereafter. The mortality from a single esophageal hemorrhage in cirrhosis is high.

For this reason they strongly advocate portal decompression as soon as varices are discovered. At present it is difficult for me to accept this approach, for the operative mortality in my experience is 14.2 per cent. Furthermore, not all patients with varices bleed, and, if they do, not all of them succumb to the first hemorrhage.

Toward the conclusion of this general analysis of but a few of the important aspects of portal decompression for variceal hemorrhage in cirrhosis, it became increasingly apparent that a number of important phases of this controversial subject have been omitted. For instance, it is well known that many patients with varices never bleed from them at all, that on occasion varices may disappear spontaneously and that in certain patients varices are not associated with portal hypertension.[33,54,55] It has further been shown that patients with varices who bleed do not necessarily do so from their varices — gastric and duodenal ulcer, acute gastritis and other common causes of upper gastrointestinal hemorrhage have all been found in the patient with cirrhosis and esophagogastric varices. Such observations as these are disturbing to those advocating portal decompression as a treatment for portal hypertension. Similarly, some effort should undoubtedly have been made to determine which ones of the several well recognized types of cirrhosis are more apt to be associated with hemorrhage, as well as which cirrhotic livers can be expected to withstand best the strain of operation or further hemorrhage. For instance, Patek et al.[25] pointed out that only 1 of 15 patients with postnecrotic cirrhosis used alcohol to excess and but 2 responded (and that only temporarily) to good dietotherapy. Certainly, there are differences in the clinical responses of patients with different types of cirrhosis. Today, these are unfortunately not well enough defined to be useful in this discussion. Perhaps it has been a serious omission to have failed to comment more extensively on the fact that the mortality of untreated cirrhosis can be materially improved by sodium restriction and prolonged dietotherapy alone. The fact that Patek and his associates[25] have been able to improve by medical means alone their over-all two-year survival by 25 per cent cannot be regarded lightly. Perhaps more should have been made of the obvious fact that large municipal hospitals draw their patients with cirrhosis from social and economic levels of society well below that of patients attracted to private hospitals. For omitting discussion of these as well as other important facets of this important problem, I can only apologize and, at the same time, express my sincere wish that neither those whose studies have been included nor those whose favorite subjects have been omitted will take offense.

TABLE 2. *Summary of 8 Patients with Variceal Hemorrhage Due to Cirrhosis Operated on as Emergency with Balloon Tamponade in Place.*

CASE NO.	SURVIVAL TO DEATH	TOTAL SURVIVAL PERIOD*	SERUM ALBUMIN	SERUM BILIRUBIN	ASCITES
	mo.	mo.	gm./100 cc.	mg./100 cc.	
Survival for more than 1 mo.:					
5	17	—	4.0	2.3	Present
7	39	—	4.1	6.0	Absent
25	6	—	3.5	2.7	Present
31	—	12	3.6	3.7	Present
35	—	9	4.8	2.8	Absent
Survival for less than 1 mo. (postoperative deaths):					
26			2.4	13.5	Absent
36			2.6	5.5	Present
48			4.3	32.1	Present

*On Jan. 1, 1955.

I am indebted to Dr. Mary Ann Payne, Dr. Ward D. O'Sullivan and the many residents who shared in the trials of these studies.

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SEGMENTAL COLITIS*

Results of Surgery

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SEGMENTAL colitis, also referred to as "regional colitis" and "right-sided colitis," has been defined as a "chronic, non-specific or cryptogenic inflammatory ulcerative or hyperplastic lesion" involving long, short or multiple segments of the colon.[1] This description requires a further clarification of the distinction between segmental colitis and the more common and discrete clinical entities of ulcerative colitis and regional enteritis.

The relation between these entities, and the line dividing them, has not always been fully agreed upon and must therefore, for purposes of presentation, be set somewhat arbitrarily. Thus, we have selected as the controlling criterion for the diagnosis of segmental colitis, inflammatory disease involving primarily the colon but sparing, initially at least, the rectum. Thirty-four patients seen at the Massachusetts General Hospital (6 males and 19 females) and the West Roxbury Veterans Administration Hospital (9 males) between 1935 and 1952 satisfied this criterion. Patients with secondary involvement of the ileum of a lesser extent than that of the colon were not excluded, nor were those whose disease later spread to the rectum. Involvement of the ileum was present in 25 (76 per cent) cases, this being a higher figure than has been reported from other studies in which a range of 18 per cent to 50 per cent has been found.[1,2] In the

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group here reported, however, the ileal disease did not in any case affect more than a few inches of the terminal ileum, nor did it approach in degree the extent of colonic involvement. Four (11.4 per cent) patients showed a normal rectal mucosa earlier in the clinical course, but involvement developed later; 24 (71.5 per cent) had no rectal involvement at any time during the illness. Included also are 6 patients (17.1 per cent) in whom a single proctoscopic examination had given equivocal evidence of rectal involvement early in the clinical course but in whom later careful examination showed the rectum to be normal.

Pathology

Although the differential diagnosis between regional ileitis and ulcerative colitis is usually clear,[2,3] in segmental colitis with ileal involvement such differentiation is not always distinct. The commonly accepted gross characteristics of all three are ulcers, pseudopolyps and other inflammatory changes (edema, granularity and easy bleeding), which may be present singly or in combination. Pathological material was available in 30 of the 34 patients studied. The 6 specimens studied from the group of 10 patients whose colons were not resected were obtained at autopsy or as a biopsy specimen at the time of the anastomotic operation. Mucosal ulcerations were found in 15 (50 per cent), pseudopolyps in 9 (30 per cent), and granuloma formation in 15 (50 per cent) of the specimens.

Table 1. *Site of Colon Involved.*

Site	No. of Patients
Ascending only	5
Ascending & transverse	9
Ascending, transverse & descending (rectum normal)	10
Transverse only	4
Descending only	1
Skip areas	5

Granuloma formation is considered to be more characteristic of regional ileitis than of ulcerative colitis. It has been reported to occur in 60 to 70 per cent of cases of the former[3] and in 4.6 per cent in cases of the latter.[4] The 50 per cent rate of granuloma in our specimens of segmental colitis is therefore higher than that in ulcerative colitis but lower than that in regional enteritis.

The segmental distribution of the lesions in the colon as determined by x-ray and operative findings is shown in Table 1.

Frequency

The relative frequency of the three diseases in a group of 858 patients seen at the Massachusetts General Hospital between 1935 and 1952 was 683 (79.6 per cent) with ulcerative colitis, 150 (17.4 per cent) with regional enteritis and 25 (3 per cent) with segmental colitis.[4] The separation of the patients into these three groups was done on both clinical and pathological grounds. The distribution corresponds roughly with that reported by others.[1,5]

Age

The mean age of the patients was twenty-eight years, the youngest being twelve and the oldest sixty-six. The peak prevalence in the third decade is characteristic of the other two diseases with which segmental colitis is compared.

Table 2. *Presenting Symptoms in 34 Cases of Segmental Colitis.*

Symptom	No. of Cases
Diarrhea	33
Weight loss	27
Blood in stools	15
Intestinal obstruction	5

Symptoms

Whereas diarrhea is a nearly universal symptom as it is in ulcerative colitis and regional ileitis, intestinal bleeding occurred in only half the cases (Table 2). This fact places it again at a midposition between ulcerative colitis, in which bleeding is common, and regional ileitis, in which it is rare. The occurrence of obstructive symptoms in only 5 (14 per cent) of the patients was unexpected and is worthy of note.

Complications

The complications listed in Table 3 re-emphasize the fact that the syndrome under discussion combines features of the other two diseases. The internal fistulas and the obstructive symptoms are similar to those found in regional enteritis, whereas the anal complications, arthritis, clubbed fingers and carcinoma, although not unknown in that disease, are more characteristic of classic ulcerative colitis, with which carcinoma has been found to occur in a range

Table 3. *Complications in 34 Cases of Segmental Colitis.*

Complication	No. of Cases
Perianal abscess	10
Anal fistula	7
Anal fissure	2
Arthritis	7
Clubbed fingers	7
Perforation with peritonitis	2
Carcinoma	2
Internal fistulas	10

of 2 per cent to 5 per cent, with an increasing prevalence beyond a ten-year duration.[6,7] Two cases of carcinoma (5.8 per cent) occurred in this series.

Medical Treatment

Because of the insecurity about the diagnosis of a lesion that involves primarily the colon, particularly regarding the possibility of carcinoma, all the patients were operated on. In none has medical therapy been carried on indefinitely. Crohn et al.[2] reported only 15.6 per cent favorable results on medical treatment. At the Mayo Clinic 50 per cent of the medically treated patients had recurrent symptoms within three years.[1]

Types and Results of Surgery

The primary purpose of this study was to evaluate the efficacy of anastomotic surgery for segmental colitis — that is, operations that by-pass or eliminate the disease without creating a colostomy or ileostomy. The operations (Table 4) performed included 23 ileocolostomies with resection of the diseased portion of the bowel, 1 segmental resection of the descending colon with colocolostomy and 10 ileocolostomies without resection.

The patients were examined one and a half to seventeen years after operation with a mean follow-up period of four and six-tenths years. The reported results vary greatly in relative length of follow-up study, and no implication of permanent cure in patients considered "well" is intended. Five of the patients had died, 2 in the immediate postoperative period (1 after ileostomy, and the other after ileosigmoidostomy, wound dehiscence and septicemia). Three died of their disease at later dates, 2 of prolonged sepsis and 1 from carcinoma of the colon. The patient who died after ileostomy was operated on for disease recurring a number of years after a previous anastomotic operation and should be classed as recurrence as should the 2 who died of prolonged sepsis. The patient who died after ileosigmoidostomy, wound dehiscence and septicemia was preoperatively considered to be in "bad condition," and the anastomotic type of operation in preference to an ileostomy in this case appears to have probably been ill advised.

At the time of follow-up examination 19 (55.8 per cent) of the patients were symptomatically well, 15 (including 1 with carcinoma) from the group of 24 who had resection of the diseased bowel and 4 from the 10 who had not. The rate of well patients in the

Table 4. *Types and Results of Surgery.*

Operation	Patients Asymptomatic without Recurrent Disease	Patients Asymptomatic with Recurrent Disease	Patients Symptomatic without Recurrent Disease	Patients Symptomatic with Recurrent Disease	Patients with Recurrence & Ileostomy but Asymptomatic*	Deaths	Cause of Death	Totals
Ileoascending colostomy	1† 0‡	0 0	0 0	0 0	0 0	0 0		1 0
Ileotransverse colostomy	3† 0‡	1 2	0 0	1 0	2 1	1 1	Disease & prolonged sepsis Disease & prolonged sepsis	8 4
Ileodescending colostomy	3†§ 1‡	0 0	0 0	0 0	0 0	1 0	Carcinoma several mo. after ileostomy	4 1
Ileosigmoidostomy	4†§ 1‡	2 0	0 0	0 0	1§ 2	0 1	Wound dehiscence with septicemia	7 4
Multiple anastomoses	0† 0‡	0 0	1 0	1 0	1 0	0 1	Unknown (after ileostomy)	3 1
Segmental resection with colonic anastomosis	1† 0‡	0 0	0 0	0 0	0 0	0 0		1 0
	14	5	1	2	7	5		34

*1 patient had annoying but not disabling ileostomy complications.
†Resection.
‡No resection.
§1 patient of each group had ileostomy closure followed by anastomotic operation.

Operation was not performed in our cases until failure of conservative management became evident except in those in which x-ray examination demonstrated a lesion in which the possibility of carcinoma could not be excluded. The shortest period of medical therapy before operation was one month, and the longest six years. The shortest duration of symptoms was three months, and the longest fifteen years. ACTH or cortisone or both were used in 6 patients. Two of them received ACTH for seven and fifteen days respectively during acute episodes of fever, diarrhea and abdominal cramps, with resultant decrease in fever and number of stools, increase in consistence of stools and subjective improvement. The other 4 were given long-term therapy over a span of several months. Temporary improvement was evident in 2 cases for only a short time before the symptoms recurred. In the other 2 steroid therapy was instituted for recurrent symptoms during the postoperative follow-up period. Both diarrhea and arthritis were relieved in 1 case, but there was no evidence of improvement in the other.

resected group was thus 62.5 per cent as contrasted to 40.0 per cent in the nonresected. The patients listed in Table 4 as asymptomatic with ileostomy are not included in the "well" group of this analysis of anastomotic operations for segmental colitis.

The follow-up picture from the viewpoint of freedom from disease was somewhat different. Five of the 19 well patients have roentgenologic or sigmoidoscopic findings consistent with mild persistent or recurrent disease, whereas 1 patient of the 15 in the "unwell" group gave no objective evidence of recurrence. In the recurrent cases the disease progressed without postoperative remission in 8; in 11 it recurred within seven to eleven months.

Ileostomy

Ileostomy was performed in 9 patients, 7 of whom are free of symptoms of colitis, although 1 of the 7 is having difficulty from complications associated with the ileostomy itself. Two died, 1 in the immediate postoperative period and the other later from carcinoma of the rectum. The former patient had previously had an ileosigmoidostomy without resection of the diseased colon. Because of extensive progressive disease, colectomy and ileostomy were performed, but the patient became a severe problem in fluid and electrolyte balance and died twenty-one days postoperatively. No patient who survived with ileostomy had difficulty with recurrent inflammatory disease.

Previous ileostomies were closed in 3 cases, in which bowel continuity was re-established by ileocolostomy, with resection of the diseased colon. Two of these (1 with an ileodescending and ileosigmoidostomy and the other with an ileosigmoidostomy and later ileostomy) have done well.

Summary and Conclusions

An analysis of 34 patients suffering from segmental colitis is presented.

Each patient had an anastomotic operation with or without resection of the involved bowel.

In 19 (55.8 per cent) of the cases the operation was successful in rendering the patient asymptomatic for an average follow-up period of four and six-tenths years. X-ray or proctoscopic evidence of mild persistent or recurrent disease was present, however, in a fourth of these asymptomatic patients. The group of asymptomatic patients included 15 (62.5 per cent) of the 24 cases with resection and 4 (40 per cent) of the 10 cases without resection of the involved bowel.

The over-all arrest rate after surgery was 44.2 per cent. Twelve (50 per cent) of the cases with resection, and 3 (30 per cent) of those without resection of the involved bowel comprise this group.

In 15 (44.2 per cent) of the patients the anastomotic operations failed. Seven (20.6 per cent of the whole group) were restored to health by ileostomy, making a current total of asymptomatic patients of 26 (76.2 per cent). Of the remaining 8 patients, 5 are dead, and 3 are living with symptoms.

In 2 patients (5.8 per cent) carcinoma of the colon developed.

Segmental colitis occupies a midposition clinically and pathologically between ulcerative colitis and regional ileitis. Surgical management is necessary in the majority of cases; an ileosigmoidostomy does not cause disability from diarrhea unless there is recurrence of the disease.

No criteria can be set forth to predict which patient will have recurrent disease after an anastomotic operation, which should therefore be attempted in most cases before a decision for ileostomy is reached.

Unless there is a definite contraindication resection of the diseased bowel should be accomplished at the same time as the anastomosis or at a planned later date.

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MULTIPLE LARGE HEMANGIOMAS OF THE LIVER*

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MULTIPLE large hemangiomas of the liver are a most unusual clinical and pathological finding. This entity must be considered when an enlarged multinodular liver is discovered so that an erroneous diagnosis of metastatic carcinoma can be avoided.

Hemangiomas, the most common benign neoplasm of the liver,[1] are localized more frequently in this organ than in any other viscera[2,3] and may be associated with angiomas in other abdominal organs and elsewhere.[4] Most hemangiomas are small and asymptomatic.[5-7] However, large hemangiomas are often clinically significant. Solitary hemangiomas are usually confined to one lobe but may extend throughout the entire liver. When hemangiomas are multiple they tend to be scattered. They may be pedunculated and may become calcified or contain phleboliths.

Shumacker[8] and Wilson and Tyson[9] have written excellent reviews on the subject of the *solitary* hemangioma of the liver. Although found at all ages, this tumor is found most often in the fourth and fifth decades and has recently been estimated to occur four and a half times as frequently in females as in males; this distribution is reversed in the older foreign literature. The initial complaint is evenly distributed between increase in size of the abdomen, vague abdominal pain and nonspecific digestive disturbances. There may be obstructive jaundice, colic, distention of the gall bladder and marked ascites.[10] The symptoms are frequently of many years' duration.

The tumor mass may attain huge size and is usually easily palpated. Occasionally, a murmur or bruit may be heard on auscultation over the mass. Spontaneous rupture is a serious complication and suggests an acute abdominal emergency such as acute appendicitis, peritonitis, perforated ulcer, acute diverticulitis or ruptured tubal pregnancy. Mechanical rupture during incision at operation or diagnostic liver biopsy has resulted in death. Excision is the treatment of choice for single tumors. X-ray therapy has been used successfully in the treatment of inoperable and multiple hemangiomas.

CASE REPORTS

CASE 1. A 63-year-old housewife was first seen in May, 1949, complaining of weight gain, poor appetite, swelling of the legs and exertional dyspnea. For several years she had noted a gradual gain of 6.8 to 9.1 kg. (15 to 20 pounds) in weight despite a decreased appetite and caloric intake. Abdominal girth had also increased during the past year. More recently mild leg edema and dyspnea on exertion had developed. A review of the systems disclosed that she had bruised easily for many years and had had frequent epistaxis during the past year.

The past and family histories were noncontributory.

Physical examination showed a sallow-complexioned average-sized woman with little panniculus and a markedly protuberant abdomen. She appeared chronically ill. The skin and mucous membranes were pale. No angiomas were noted. The right leaf of the diaphragm was elevated and moved poorly on inspiration. There were many dilated superficial veins on the skin of the abdomen. A small amount of ascites was present. The liver edge was palpated 16 cm. below the right costal margin and 20 cm. below the left costal margin in the midclavicular lines. The liver was hard and nontender. Many nodules of various sizes were palpated over the entire surface. One nodule at the inferior edge of the right lobe measured 7 by 6 cm. The tip of the spleen extended 2 fingerbreadths beneath the left costal margin. There was minimal bilateral pretibial edema.

Examination of the blood revealed a red-cell count of 3,840,000, with a hemoglobin of 9.5 gm. per 100 cc., and a white-cell count of 4800, with a normal differential. Ample platelets were noted on a blood smear. The urine was normal.

The clinical diagnosis was far-advanced metastatic carcinoma of the liver that would soon prove to be fatal. There was thought to be little or no point in searching for the primary site.

The patient was placed on a high-carbohydrate-high-protein, low-fat and low-salt diet, diuretics and iron preparations. Little change occurred during the following year except for an increase in ascites. She was hospitalized, and an abdominal paracentesis was performed. What were considered "adenocarcinomatous cells" were recovered from a cell block of the ascitic fluid. Multiple liver-function tests were within normal limits. A liver biopsy revealed "normal liver tissue." The diagnosis of metastatic carcinoma of the liver, primary site unknown, was made again.

The clinical condition remained stationary during the ensuing 4½ years except for a rapid reaccumulation of ascites after each paracentesis. There were many admissions to different hospitals at intervals of 2 or 3 months for abdominal paracentesis necessitated by abdominal discomfort and severe dyspnea. At each tap serosanguineous fluid was removed with a loss of 11.3 to 18.1 kg. (25 to 40 pounds). Liver-function tests, cell blocks of the ascitic fluid and other studies performed on these admissions failed to uncover the etiology of the disease. Carcinoma cells were never again discovered in the ascitic fluid. No evidence of esophageal varices could be demonstrated on x-ray examination. A complete gastrointestinal x-ray survey did not give evidence of neoplasm; the duodenal bulb and 1st portion of the duodenum were displaced forward, suggesting the presence of a retroperitoneal mass. No other symptoms developed, and the patient remained in generally fair health, being able to carry on her household chores. After a paracentesis in June, 1954, a venous hum was heard in the region of the umbilicus.

In January, 1954, an exploratory laparotomy was performed to establish a definite diagnosis. The liver was huge and extended well below the umbilicus. It was firm and paler than normal, and over its surface were numerous protrusions of very vascular tissue. These appeared to be hemangiomas, and on compression blood could be expressed from them so that they became almost flat and level with the surface of the liver. Biopsy of 2 of these lesions was performed. The spleen was soft, about twice its normal size and pushed posteriorly. General examination of the abdominal cavity gave no evidence of tumor in any of the viscera.

On microscopical examination large foci of dilated capil-

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laries replaced normal liver tissue. The supporting stroma of these capillaries consisted of a hyalinized collagenous mass. Large bands of fibrous tissue in the periportal area caused a lobulation of the liver parenchyma (Fig. 1). The liver cells in the region of the central veins were hyalinized. The diagnosis was hemangioma and cirrhosis of the liver.

Postoperatively the patient received x-ray therapy to the liver. Although some decrease in the size of the liver followed this treatment the ascitic fluid reaccumulated more rapidly than before.

Case 2. A 33-year-old man had first been seen in January, 1934, at the age of 13, complaining of paroxysmal epigastric pain. The history of the present illness dated back 1 year, during which there were repeated episodes of epigastric pain associated with nausea and vomiting. The pain was unrelated to meals. He had lost 4.5 kg. (10 pounds) in weight and was listless and fatigued.

The past and family histories and a review of the systems were noncontributory.

Physical examination disclosed fullness in the right upper quadrant and epigastrium. A large, firm, rounded, slightly tender mass that moved with respiration was palpated below the right costal margin. It descended to 1 cm. below the umbilicus on full inspiration. The spleen was not palpable.

Examination of the blood showed a red-cell count of 3,160,000, with a hemoglobin of 9.6 gm. per 100 cc., and a white-cell count of 14,150, with a normal differential. The icteric index was 14.8 units. The total protein was 7.54 gm., the albumin 3.76 gm., and the globulin 3.78 gm. per 100 cc. Other liver-function tests were within normal limits.

The temperature ranged between 100 and 103°F. The clinical diagnosis was an acute surgical condition of the abdomen. An exploratory laparotomy was performed. At operation the right lobe of the liver was enlarged to 3 times its normal size and occupied the entire right hypochondrium, subphrenic and epigastric regions. It was fluctuant; the left lobe was 1½ times its normal size and was firmer in consistence. A biopsy was performed. Microscopical examination showed numerous endothelial-lined spaces replacing normal liver tissue. Adjacent to these spaces were fragments of liver tissue that gave signs of compression. The diagnosis was hemangioma of the liver.

Until 1952 the patient received many courses of x-ray therapy, which resulted in a decrease in the size of the liver. He still complains of intermittent dull pain in the right upper quadrant. Recently, x-ray study of the abdomen showed numerous discrete areas of calcification throughout the liver. In September, 1954, multiple liver-function tests were negative. There is no demonstrable evidence of portal hypertension.

Summary

Two cases of multiple hemangiomas of the liver are described. Because of a multinodular liver on examination, recurrent ascites and a report of "cancer" cells in one sample of ascitic fluid in 1 case, it was assumed that the patient had a metastatic carcinoma of the liver from an undiscoverable primary site. Normal liver tissue was found on three liver biopsies. In view of the actual disease it was most fortunate that the tumors were never punctured during the needling, for the patient might have died from uncontrollable hemorrhage. After many years of symptoms and five years of medical observation, a diagnosis of multiple benign hemangiomas of the liver was finally established after an exploratory laparotomy. X-ray therapy was unsuccessful in obliteration of the hemangiomas and in reducing the rapidity of accumulation of ascites.

The onset of symptoms in the other case suggested an acute surgical condition of the abdomen. Exploration demonstrated the true nature early in the clinical course. X-ray therapy was successful in treatment. During more than twenty years of observation there have been only minimal symptoms without evidence of hepatic dysfunction or portal hypertension.

Figure 1. *Biopsy Specimen of the Liver Obtained at Exploratory Laparotomy in Case 1 (Hematoxylin and Eosin Stain X85).*

The tumor consists of vascular channels supported by abundant dense fibrous tissue. Adjoining liver tissue is seen.

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MEDICAL PROGRESS

APPLICATION OF HOMEOSTATIC PRINCIPLES TO THE PRACTICE OF PARENTERAL FLUID THERAPY*

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THE comments on parenteral fluid therapy that follow are based on the fact that there are limits to the body's capacity to conserve and to eliminate, in accordance with homeostatic needs, water and solutes given either orally or parenterally.[1,2] For each substance there is a *physiologic minimum requirement*, which is the least intake of the substance in question needed to balance output and hence to prevent deficits when conservation forces are acting maximally. There is also for each substance a *physiologic maximum tolerance*, defined as the largest amount of the substance that can be taken and eliminated without seriously disturbing body composition.

Rates of water and solute administration that fall between the limits defined above may be said to be within the *safe working ranges* of a person's homeostatic function. Such rates of administration give the patient an opportunity to maintain the *status quo* by eliminating the substance given, to repair deficits by retaining a part or all of the amount represented by the difference between that given and the physiologic minimum or to eliminate bodily surplus by excreting the substance more rapidly than the rate at which it is being given. Administration of substances either orally or parenterally at rates that fall outside the homeostatic limits described above result in abnormal changes of the types listed in Table 1.

The safe working range defined by the minimum requirement and maximum tolerance for each substance expressed per square meter of body surface area per unit of time is fairly constant for normal persons, but may be reduced in disease. Reductions may be temporary and functional or the permanent consequence of organic disorders involving one or more of the systems concerned with body homeostasis. When such a disturbance is present it is particularly important to relate rates of administration to limits of excretory capacity.

In the following section, the physiologic characteristics, conditions appropriate for use and tolerable doses and rates of administration of a number of the solutions commonly used in parenteral fluid therapy are discussed. The suggestions made are to be considered approximate guides to therapy. Successful utilization of these guides depends on careful appraisal of the status and of the particular therapeutic needs of each patient. To permit the application of therapeutic considerations to patients varying in size from the premature infant to the adult, most of the values are expressed in terms of square meter of body surface per unit of time.[3] The approximate surface area of patients of various weights may be estimated with the aid of Table 2.

The final section of the paper presents sample studies of metabolic balance illustrating the manner in which patients respond when given parenteral fluid therapy that falls within the safe range of homeostatic function.

CHARACTERISTICS, INDICATIONS AND ADMINISTRATION OF COMMONLY USED FLUIDS

Dextrose in Water Solution

Physiologic characteristics. Because 5 per cent dextrose in water contains 280 milliosmols of dextrose per liter it is approximately isotonic with plasma at the time of administration. On infusion, however, the dextrose of the solution is stored as glycogen or fat or metabolized to carbon dioxide (most of which is excreted by way of the lungs) leaving solute-free water. The body needs and can utilize a limited amount of such "free" water to cover insensible water losses, the formation of sweat, the excretion of endogenous waste products by way of the urine and the correction of water deficits or hypohydration.‖[1] Because this electrolyte free water is completely lacking in the major solutes essential to cellular and extracellular body fluids, it is ineffective in repairing solute deficits. Moreover, dextrose in water contains no solutes that the organism can use for urine formation. In conservative terms, a minimum of 1 milliosmol of electrolyte, nitrogenous waste product or other solute must be available to accompany each 10 ml. of urine water.**[4] Persons taking an ordinary diet excrete about 600 milliosmols of solute per

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‖Hypohydration is intended to signify lack of water relative to solutes (cubic centimeters per milliosmol subnormal) in body fluids.

**The majority of healthy persons can secrete urine in which there is more than 10 ml. of water per milliosmol of total solute. However, none will be jeopardized by arbitrarily setting the limit at this point where a few have reached their maximal water excretory capacity whereas 50 per cent would be expected to suffer water retention if the upper limit of water excretory capacity were set at a larger figure more truly representative of average maximum excretory capacity.

square meter of body surface per day. When a person is receiving only dextrose in water, solute output may amount to only 200 milliosmols, which will limit maximum urine output to but 2000 ml. per square meter per day.

Conditions in which it may be appropriate to use dextrose in water. Generally speaking, these conditions are characterized by an acute or subacute need for "free" water and sugar in the presence of greatly reduced tolerance or temporary lack of need for electrolytes or other fixed solute. The following conditions are illustrative of these situations.

1. *Renal failure with severe oliguria or anuria not due to body electrolyte or water deficiency.* Such failure may occur in acute hemorrhagic nephritis, renal drug and transfusion reactions, chronic nephritis or congestive heart failure. Here dextrose in water, without adding to fixed solute stores in the body, provides water needed to compensate for that being lost via the lungs and skin and dextrose that acts to suppress ketosis and to reduce tissue protoplasmic catabolism to minimum levels.*

TABLE 1. *Effects of Administering Too Little or Too Much Water and Certain Solutes.**

SUBSTANCE	AMOUNT ADMINISTERED	
	TOO LITTLE	TOO MUCH
Water	A. Serum solute & sodium concentration increased Highly concentrated urine Thirst Oliguria Fever Circulatory failure	B. Serum solute & sodium concentration decreased Very dilute urine Polyuria Intracranial hypertension Headache, confusion, nausea & vomiting Weakness Muscle twitchings & cramps Convulsions Coma
Sodium	C. Extracellular fluid volume decreased Hemoconcentration Lost tissue elasticity Microcardia Hypotension Circulatory failure Uremia	D. Extracellular fluid volume increased Edema formation & congestive heart failure Tendency to potassium deficiency
Potassium	E. Apathy Lethargy Muscle weakness Electrocardiographic changes Ileus or diarrhea Hypopotassemia Metabolic alkalosis	F. Hyperpotassemia Electrocardiographic changes Muscle weakness Cardiac arrest
Phosphorus	G. Hypophosphatemia ?Other effects	H. Hyperphosphatemia Hypocalcemia Tetany
Carbohydrate	I. Ketosis Protoplasmic catabolism augmented Tendency to greater water & electrolyte loses	J. Hyperglycemia Glycosuria Hepatic failure

*Adapted from Talbot et al.[1]

2. *Diabetes insipidus, uncontrolled by antidiuretic hormone.* Here, the need is apt to be for large volumes of water free from electrolytes since urinary solutes may require water in a ratio of between 10 and 20 ml. per milliosmol of solute (normal, about 1 ml.) for elimination.

3. *Intraoperative and first postoperative day in patients who are free from metabolic disturbances and not suffering abnormal fluid or electrolyte losses and who may be expected to take fluids by mouth within a few hours.* In such patients the need is chiefly for "free" water and sugar. Hence, dextrose in water may be considered satisfactory provided the rate of administration is kept within the specific limits of tolerance outlined below and provided it is not continued for more than a few hours.

TABLE 2. *Body-Weight and Surface-Area Equivalents.**

WEIGHT		SURFACE AREA
lb.	*kg.*	*sq. meters*
4.4	2	0.15
11.0	5	0.25
22.0	10	0.45
33.0	15	0.60
44.0	20	0.80
66.0	30	1.05
88.0	40	1.30
110.0	50	1.50
132.0	60	1.65
154.0	70	1.75
176.0	80	1.85
198.0	90	1.95
220.0	100	2.05

*Adapted from Crawford et al.[3]

Tolerable doses and rates of administration.

1. *For patients with normal homeostatic systems who are not under the influence of ether, morphine and related drugs.* The physiologic minimum requirement is 900 ml. of 5 or 10 per cent dextrose in water solution per square meter of body surface per day. The physiologic maximum tolerance is 2700 ml. of 5 or 10 per cent dextrose in water solution per square meter per day. To avoid undue fluctuations in body water and electrolyte content and prolonged intervals of fasting, the total day's allotment should be spread out over a period of more than twelve hours or given in two divided doses over periods lasting at least three hours each.

Specifically excluded from this group are persons under the influence of ether, morphine and related drugs and those with congestive heart failure, severe hepatic or renal impairment or diabetes insipidus. The special needs of patients with these conditions are indicated in approximate terms in the following paragraphs.

2. *For patients with conditions that reduce neuro-*

*A minimum of 75 gm. of dextrose per square meter of body surface per day is needed to eliminate ketosis and reduce protoplasmic catabolism to approximately minimum levels.[2] This means that it is desirable to use 10 per cent dextrose solution when minimum rates of infusion are in effect.

hypophyseal-renal capacity to maintain body water concentration homeostasis (Table 3).

Anuric patients have no capacity to adjust water output in accordance with homeostatic needs. It is therefore imperative to relate water input much more carefully to insensible water output than is necessary in other patients. This accounts for the fact that infants, children and adults are listed separately in Table 3. For each of these age groups the value given is equal to average extrarenal loss for age,[5] less an average value for water of oxidation and tissue catabolism (250 ml. per square meter of body surface per day). The anuric patient given dextrose in water in amounts just sufficient to maintain body water concentration at normal levels should lose body weight as he oxidizes body fat at a rate of at least 150 gm. per square meter per day. Rates of catabolism considerably in excess of this may occur in the anuric patient.[6]

TABLE 3. *Requirements for Patients with Conditions Reducing Neurohypophyseal-Renal Capacity to Maintain Body-Water-Concentration Homeostasis.*

CONDITION	AGE OF PATIENT	STRENGTH OF DEXTROSE IN WATER SOLUTION*	APPROXIMATE DOSE	TIME FOR ADMINISTRATION
	yr.	gm./100 ml.	ml./sq.m./day	hr.
Anuria	<2	10	1000	At least 12
	2-8	10	700	At least 12
	>8	15-25	400	At least 12
Obligatory oliguria due to ether or morphine, congestive heart failure, hepatic disease, advanced nephritis & acute hemorrhagic nephritis	<2	5	1400	At least 12
	2-8	5	1100	At least 12
	>8	10	800	At least 12
Diabetes insipidus uncontrolled by antidiuretic hormone	All ages	5	2500-4000	24

*Concentration of dextrose adjusted to provide patients receiving very limited volumes of water with minimum of about 75 gm. of carbohydrate per square meter per day.

In patients with obligatory oliguria secondary to loss of ability to increase urinary water concentration, the need is equal to the extrarenal loss as for anuric patients in addition to about 2 ml. per milliosmol of solute excreted by way of the urine. In patients given dextrose in water this additional need amounts to about 200 milliosmols X 2 ml., or approximately 400 ml. per square meter of body surface per day.

In uncontrolled diabetes insipidus the requirements are the same as those for anuric patients in addition to between 10 and 15 ml. per milliosmol of urinary solute, which, at 200 milliosmols per square meter per day, amounts to between 2000 and 3000 ml. per square meter per day. These patients can usually vary their urine water concentration within limits sufficiently broad to permit use of the same water-allowance figure for patients of all ages.

0.85 Per Cent Saline in 5 Per Cent Dextrose Solution

Physiologic characteristics. The dextrose of this solution serves as described above to suppress ketosis and minimize protoplasmic catabolism. Though an effective solute at the time of administration,* in the absence of uncontrolled diabetes mellitus, dextrose is rapidly metabolized and disappears as a solute. Hence it is excluded from the following discussions of relations between water and "fixed" solute of parenteral fluid solutions.

The sodium concentration (150 milliequiv. per liter) of the saline in dextrose solution is close to that of extracellular water (about 140 milliequiv. per liter). On the other hand the chloride concentration (150 milliequiv. per liter) is about 30 per cent greater than that normally found in extracellular water (about 115 milliequiv. per liter). Under circumstances in which the body is unable to eliminate this excess chloride as rapidly as it is infused, the saline in dextrose solution tends to produce a rise in plasma chloride concentration and hence acidosis. This effect can be eliminated by substitution of an appropriate amount of sodium bicarbonate or lactate for sodium chloride as described below under dextrose-saline-lactate solution.

Saline in dextrose, like other solutions of isotonic total inorganic solute content, is of low free-water content. In this fluid, as in normal extracellular fluid, there is about 3.5 ml. of water per milliosmol of fixed solute. Accordingly, if the sodium and chloride are retained by the body to make up volume deficits of extracellular solute, all the water of the solution is needed for the formation of isotonic body fluid. If any of the sodium or chloride in the solution is eliminated by way of the kidneys, in normally functioning persons at least 0.7 ml. and, in chronically stressed persons, as much as 2.5 ml. of water may be needed to accompany each milliosmol of sodium and chloride appearing in the urine. If the 300 milliosmols of sodium and chloride contained in a liter of the solution is excreted in company with 2.5 ml. of water, 750 ml. of water is necessary for urinary transport, leaving only 250 ml. of free water available to cover insensible water losses and water required for renal excretion of endogenous waste products.

Attempts to overcome this free-water deficiency by an increase in the volume infused are not apt to yield satisfactory results. If one were to give the largest quantity of sodium that the body can be depended on to eliminate reasonably promptly if unneeded (that is, about 225 milliequiv. of sodium per square meter of body surface per day, or the quantity contained in 1500 ml. of saline in dextrose solution[1]) one would be providing the stressed patient with a free-water

*As administered, the solution has twice the tonicity of normal plasma. This renders it unsuitable for subcutaneous administration.

allotment of about 400 ml. per square meter per day. This amount is less than half that required to equalize water lost insensibly (average, all ages, about 1000 per square meter per day) and used in the renal elimination of a minimum load of endogenous solutes (200 milliosmols per square meter per day at at least 1 ml. per milliosmol). The administration of a larger volume of solution is likely to result in obligatory overexpansion of body sodium stores, with resultant edema formation (Table 1).

These shortcomings of the solution can be reduced by dilution with an equal volume of dextrose in water. The resultant solution contains 75 milliequiv. of sodium, 75 milliequiv. of chloride and 50 gm. of dextrose per liter and has a ratio of water to inorganic solute of about 7 ml. per milliosmol. This solution contains not only much more free water than the undiluted saline in dextrose mixture, but also twice as much (up to 3000 ml. per square meter per day) can be given before the maximum tolerable dose of sodium (225 milliequiv. per square meter per day) is reached.

Saline in dextrose solutions, being totally lacking in potassium, fail to provide the body with an ingredient essential to the repair of cellular electrolyte deficits. Moreover, the administration of saline solutions in the absence of potassium is apt to accentuate the degree of potassium deficiency whose various manifestations are outlined in Table 1. Accordingly, in persons with normally functioning homeostatic systems, solutions containing both potassium and sodium (described in a later section under "multiple electrolyte solution") are usually considered preferable to saline in dextrose for maintenance and repair.

Conditions in which it may be appropriate to use saline in dextrose (saline lactate, described below, is preferable to saline solution for many patients). Appropriate use of saline in dextrose is limited to situations in which there is an emergency need to expand extracellular fluid to sustain vascular volume. Thus, the solution may be of use in the treatment of circulatory insufficiency and shock *due to primary hypovolemia*. The therapy of shock due to primary loss of vascular tone should emphasize restoration of the capacity of the vascular system. Administration of the mixture greatly to expand vascular volume is of dubious value and may actually do harm.

Together with blood or plasma or both, saline in dextrose may be needed in the early hours of therapy of patients with extensive burns. Such patients tend to become hypovolemic because water, protein and electrolytes leak out of the vascular compartment as a result of thermal injury. In such cases as in any others in which sodium salt solutions are used for special purposes, it is essential to dilute the isotonic saline in dextrose with dextrose in water in amounts sufficient to cover insensible water losses and to provide free water for the urinary excretion of solutes.

Tolerable doses and rates of administration.

1. *For circulatory failure due to hypovolemia in patients without burns and with basically normal homeostatic system.* The doses and rates are shown in Table 4. Because hypovolemia is a phenomenon related to mass, total dose is estimated as 3 to 5 per cent of body weight, or 10 to 20 per cent of normal extracellular-fluid volume. If this volume or a lesser amount serves to restore circulatory efficiency to the extent that the pulse and blood pressure are satisfactory, signs of shock are absent and evidence of urine flow is present, it is appropriate to consider use of a multiple electrolyte solution to provide for residual maintenance and repair needs as described below. If, on the other hand, the dose of saline in dextrose outlined above fails to restore circulatory efficiency, the diagnosis and therapeutic needs should be reviewed, for it is likely that the administration of larger doses without clear, specific indication will do more harm than good.

TABLE 4. *Tolerable Doses and Rates of Administration of Saline in Dextrose Solution.*

AVERAGE (ALL PERSONS)		ADULT	
RATE OF ADMINISTRATION	TOTAL DOSE	RATE OF ADMINISTRATION	TOTAL DOSE
ml./sq.m./min.	ml./kg.	ml./min.	ml./patient
8-10	30-50	15-20	2000-3500

2. *For patients with extensive burns.* These patients constitute a special group with unusual needs for saline solution because thermal injury leads to seepage of large quantities of water, protein and extracellular electrolytes from the vascular compartment into the burned areas. The volumes of plasma in addition to either saline or saline lactate solution (as described below) presented in Table 5 usually suffice to compensate for such dislocations and losses of fluids in the first twenty-four hours after the accident.[7,8*] Half the total is given in the first eight, and the other half in the next sixteen hours. In the second day of therapy it should not be necessary to give more than half the first day's allotment, and it may not be necessary to give any of these solutions.

During each twenty-four hours, between 1200 and 1500 ml. of dextrose in water per square meter of body surface must be added to the fluids described above to cover insensible losses of water via lungs and skin and to provide water for renal elimination of the heavy solute loads resultant from tissue in-

*The figures listed should be considered as rough guides only. In providing for the needs of extensively burned patients, the adequacy of the allowances should be checked by frequent measurements of rate of urine flow and of the hematocrit or blood hemoglobin levels. The occurrence of urine flows in excess of 30 ml. per square meter of body surface per hour, of a hematocrit reading below 40 per cent or of a hemoglobin concentration below 11 gm. per 100 ml. is considered indicative of excessive fluid administration. Contrariwise, rates of urine flow below 10 ml. per square meter per hour, hematocrit readings above 50 per cent and hemoglobin levels above 15 gm. per 100 ml. may be considered indicative of inadequate fluid therapy.[8,9]

jury. This dextrose in water should be mixed with the total daily allotment of plasma and other isotonic electrolyte solution. After forty-eight hours intravenous administration of fluids should be limited to basic multiple electrolyte and dextrose maintenance therapy (as described below). Parenteral fluids should be eliminated entirely as soon as the patient is able to take adequate fluids by mouth.

Saline Lactate in 5 Per Cent Dextrose Solution

Composition. The solution contains 140 milliequiv. of sodium, 110 milliequiv. of chloride, 30 milliequiv. of lactate (that is, potential bicarbonate) and 50 gm. of dextrose per liter.*

TABLE 5. *Requirements of Saline (or Saline Lactate) in Dextrose for Patients with Extensive Burns.*

FLUID	TOTAL DOSAGE PER 1% OF SURFACE AREA BURNED
	ml./sq.m.
Plasma*	60
Saline (or saline lactate) in dextrose	20
Total	80

*Assumed that in course of preparation, plasma has been diluted approximately 30 per cent by addition of sodium citrate & citric acid, anticoagulant mixture (about 20 per cent by volume) and of concentrated dextrose (about 10 per cent by volume).

Physiologic characteristics. This solution is representative of many saline-bicarbonate and saline-lactate mixtures that have been prepared in an effort to modify saline in dextrose so that it contains chloride and bicarbonate as well as sodium at concentrations corresponding closely to those found in normal extracellular water (140 milliequiv. of sodium, 115 milliequiv. of chloride and 27 milliequiv. of potential bicarbonate per liter). The present solution may therefore be considered more nearly physiologically neutral in terms of extracellular fluid than isotonic saline solution, with its excess of chloride and lack of bicarbonate or potential bicarbonate. Like saline in dextrose it lacks cellular electrolytes and provides the body with little and at times no free water to cover insensible losses, to provide for the urinary excretion of endogenous solutes or to repair hypohydration. As with saline in dextrose, this water deficiency can be reduced by dilution of saline-lactate-dextrose solution with an equal amount of dextrose in water to make a solution containing per liter 70 milliequiv. of sodium, 55 milliequiv. of chloride and 15 milliequiv. of lactate or bicarbonate.†

Conditions in which it may be appropriate to use saline-lactate-dextrose solution. This solution is considered preferable to saline in dextrose for nearly every condition in which there is an emergency need to expand extracellular fluid to sustain vascular volume. An exception is severe hypochloremic alkalosis of the type seen most frequently after obstruction of the upper intestine, with extensive vomiting of gastric hydrochloric acid. Although the solution may be of great value in the initial hydration of severely dehydrated patients, it should not be used for more than a limited period, as outlined above, unless the patient has a particular need for sodium in company with intolerance for potassium. This situation may be encountered early in the treatment of extensively burned patients and in those with adrenocortical failure (Addison's disease) or advanced nephritis.‡ When it is to be given to such persons for special maintenance as well as initial restorative purposes, saline lactate in dextrose should be at least partly diluted with dextrose in water as mentioned above.

Tolerable doses and rates of administration. These are the same as those outlined above for saline in dextrose.

Multiple Electrolyte Solution in 5 Per Cent Dextrose

Composition. This solution contains 40 milliequiv. of sodium, 35 milliequiv. of potassium, 40 milliequiv. of chloride, 20 milliequiv. of lactate, 15 milliequiv. of phosphate and 50 gm. of dextrose per liter.§

Physiologic characteristics. The solution is designed to provide the organism with water, carbohydrate and the chief cellular and extracellular electrolytes in about equally tolerable amounts.[1,11] As infused, the solution has one and a half times the tonicity of plasma; two thirds of this tonicity is referable to the 5 per cent dextrose and a third to fixed electrolyte solutes. As mentioned above under dextrose in water the dextrose is eliminated as a solute shortly after it is introduced into the circulation. When the osmotic activity due to dextrose has been eliminated the solution has a residual tonicity of 150 milliosmols per liter. This means that the solution provides a considerable amount of free water, which can be used as needed to cover insensible water losses, urinary excretion of endogenous waste products and repair of hypohydration.

In the determining of the relative concentrations of sodium and potassium in this solution, recognition was given to the fact that depleted patients may need either considerable amounts or none of either one or both of these electrolytes. It also appears that the ranges of bodily capacity to retain and reject sodium and potassium in accordance with homeostatic needs are approximately equal. Taken together, these two factors made it appear appropriate to have the solution contain these two cations in approximately equivalent amounts. In considering the absolute amounts of these cations in the solu-

*A liter of this solution can be prepared by mixture of 30 ml. of molar sodium lactate, 750 ml. of 5 per cent saline in dextrose and 220 ml. of 5 per cent dextrose in water.

†A closely similar solution is commercially available from Mead Johnson and Company, Evansville, Indiana.

‡Even these patients may need potassium as well as sodium for repair and maintenance purposes.

§This solution is commercially available from Mead Johnson and Company, Evansville, Indiana.

tion, attention has been given to making them available to the organism in company with appreciable and yet physiologically tolerable amounts of free as well as of solute-bound or obligated water.

Similar principles were used in determining the chloride, lactate and phosphate content of the solution. Consequently, when the solution is given at rates that provide sodium and potassium at physiologically tolerable levels, it also delivers chloride, lactate and phosphate at rates that lie within their respective safe working ranges. Although phosphate is potentially available to meet essential body fluid needs because there are large skeletal stores of this anion, its inclusion makes it easily available for repair of cellular deficits and serves to keep the concentrations of chloride and lactate in proportion to their companion cation, sodium. Moreover, unneeded phosphate, when excreted by way of the kidneys, increases the urinary buffer capacity.[10]

Conditions in which it may be appropriate to use multiple electrolyte solution in dextrose. This solution is suitable for maintenance and repair in patients whose neurohypophyseal, parathyroid, adrenocortical and renocardiovascular systems are functionally intact. Circulatory failure due to loss of body solutes and water (hypovolemia) may lead temporarily to intolerance for potassium. Accordingly, it is recommended that therapy with multiple electrolyte solution for patients with circulatory failure be postponed until circulatory efficiency has been restored by administration of sodium salt solution as outlined in the first two sections of this report. Just before institution of multiple electrolyte solution, it is often advisable to check the patient's potassium status by means of electrocardiography.* In persons who are free from heart disease and are not receiving digitalis, the occurrence of abnormally peaked T waves in Lead 2† (Fig. 1) ordinarily indicates the presence of excess potassium in the circulation or myocardium and hence the need to postpone administration of this electrolyte. Under conditions of renal failure, hyperkalemia also constitutes a contraindication to potassium administration.

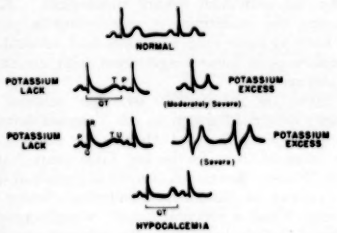

FIGURE 1. *Representative Lead 2 Electrocardiographic Tracings, Illustrating the Flattening of the T Waves and Prolongation of the QT Interval That Tend to Become Evident in Potassium Insufficiency, the Peaking of the T Waves and Disorganization of the QRS Complex Seen in Potassium Intoxication and the Prolongation of the QT Interval without the Changes in Wave Shape Seen in Simple Hypocalcemia.*

Patients with permanent, severe disability of one or more of the systems mentioned above need therapy that is accurately tailored to their particular homeostatic needs. Therefore, this solution should not be given to such persons unless it happens to fit their specific requirements satisfactorily.

TABLE 6. *Doses and Rates of Administration of Multiple Electrolyte Solutions.*

TYPE OF NEED	AMOUNT	TIME FOR INFUSION
	ml./sq.m./ 24 hr.	
Simple maintenance	1500	At least 12 hr. or 2 3-hr. periods at 12-hr. intervals
Normal maintenance & abnormal maintenance & repair	2000-4000	Preferably 24 hr.

Doses and rates of administration. These vary according to whether the patient simply needs to maintain a satisfactory *status quo* or whether he is depleted or suffering continued abnormal losses (Table 6).

The "simple-maintenance" allotment provides water, sugar and electrolytes in amounts only slightly in excess of physiologic minimum requirements. This rate corresponds closely to that indicated for patients with temporary obligatory oliguria due to ether or morphine and its derivatives (as pointed out in the first section under "dextrose in water").

It is intended that the amount for normal maintenance in addition to abnormal maintenance or repair should be enough larger than the physiologic minimum figure to permit the patient to cover his maintenance requirements and in addition to correct deficits at a reasonably satisfactory rate. Ordinarily, it is not necessary to give more than 3500 ml. per square meter of body surface per day to satisfy the over-all requirements of patients suffering deficits or continuing abnormal losses or both.

(To be concluded)

*Since some severely depleted patients may be in urgent need of potassium as well as other substances at the very outset of therapy, it may also be advantageous to obtain an electrocardiogram at the time of the initial appraisal of the patient. Delay can be eliminated by use of the direct-writing type of electrocardiographic instrument.[12]

†It is important to remember that acidosis may be associated with elevation of the T wave. In acidosis, however, the T waves appear domed rather than peaked.

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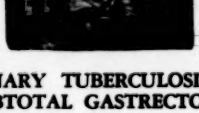
MEDICAL INTELLIGENCE



PULMONARY TUBERCULOSIS AFTER SUBTOTAL GASTRECTOMY

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IN a large tuberculosis hospital (615 beds) I have been impressed by the number of patients who have been admitted in the last few years with active pulmonary tuberculosis after subtotal gastrectomy for peptic ulcer.

One of the chief factors leading to this chronic infection is lack of resistance. Starvation, faulty assimilation of ingested food, metabolic imbalance, long-continued stress and poor hygienic surroundings seem to favor the development of tuberculosis.

When, as clinical director of the United States Navy Hospital Ship, *Benevolence*, it was my privilege to assist in screening 1500 recently liberated prisoners of war in Japan at the surrender in 1945, I was impressed with the amount and type of pulmonary tuberculosis in these half-starved prisoners. The disease was pneumonic in many, and extensive and widely distributed. Large lower-lobe tension cavities were seen in many, with or without the more commonly observed apical and subapical disease. This is the type of tuberculosis often seen in patients with severe uncontrolled diabetes and in Negroes, whose resistance to the disease has been believed to be somewhat below that of the white race. So it has seemed that the faulty assimilation of the proper food elements, after an operation such as subtotal gastrectomy, sets up a chain of events completely foreign to the natural function of the gastrointestinal tract, that a state of malnutrition is the rule, rather than the exception, and that a logical sequel would be lowered resistance with the acquirement of tuberculosis.

The literature is scanty on the subject, the European literature[1-4] exceeding that of this country. Foreign investigators agree in the assumption that subtotal gastrectomy has an adverse effect on inactive tuberculosis, according to Warthin, who reviewed the foreign articles. Warthin,[5] in 1953, reported 7 cases of active pulmonary tuberculosis that constituted approximately 2 per cent of 356 patients with peptic ulcer treated by gastrectomy. His cases represented reactivations of old healed tuberculous foci, for the most part, and the reactivations were notably of the pneumonic type, with an over-all mortality of 25 per cent. The description of these cases seemed to fit with that of the prisoners of war described above. He did not attempt to define the mechanism in the breakdown of immunity in these patients but suggested that: "loss of weight as well as stress somehow predisposes an individual toward tuberculosis." King,[6] discussing the occurrence of tuberculosis in persons who have at some time previously had subtotal gastrectomies, is in general agreement with me on the probable causes.

A review of the patients who had subtotal gastrectomy before admission to the Veterans Administration Hospital, Rutland Heights, in the last few years, most of them in the last three years, has revealed 21 cases. Seventeen of these patients had never had evidence of pulmonary tuberculosis before gastrectomy, 4 had a previous history of pulmonary tuberculosis, which in all cases had been considered inactive and which had been reactivated after gastrectomy. The age range was between thirty-two and sixty-six years; all were men, and all had advanced disease, some cases being pneumonic tuberculosis. In this group of 21 patients there were 2 deaths, but all patients in the group had the distinct advantage of receiving the important adjunct to bed care, the antituberculosis drugs (streptomycin, dihydrostreptomycin, isoniazid and para-aminosalicylic acid in various combinations), and this accounts chiefly for the relatively low death rate in a group with such low resistance and advanced disease. Table 1 reveals the time of operation and time of diagnosis of active pulmonary tuberculosis. The interval between operation and diagnosis of tuberculosis ranged from the extremes of fourteen days to fourteen years, the average being four years.

DISCUSSION

Certainly, cause and effect could not be held responsible for the two extremes in interval between operation and diagnosis of tuberculosis. The case diagnosed as active tuberculosis two weeks after gas-

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trectomy was presumably active at operation, although it had been considered inactive at that time. The patient in whom tuberculosis did not develop until fourteen years after gastrectomy took an unusual time to show the results of low resistance. However, Table 1 shows that, in most cases, two, three or four years elapsed between operation and diagnosis of tuberculosis. Even this period seems long, but several factors may determine the time in which tuberculosis may put in its appearance: the interval between gastrectomy and the time when the patient comes in contact with an open case of tuberculosis and thereby acquires the infection by contact; the time after infection is acquired before a correct diagnosis of tuberculosis is made; and the time after gastrectomy for a patient's resistance to become so low that he may, when in contact with infection, acquire it. It can be clearly appreciated that after gastrectomy a patient may be confined in a home or rural community for many months or even years and that contact with an open case may be unlikely. Thus, in spite of low resistance, he does not acquire tuberculosis since he has no contact with it. It can likewise be appreciated that the usual signs and symptoms of oncoming tuberculosis may be misinterpreted by family and physician as part of the picture of convalescence from the gastrectomy. Since the disease is not suspected no x-ray examination of the chest is made, and the diagnosis of active disease may be delayed for months.

TABLE 1. *Cases of Active Pulmonary Tuberculosis after Gastrectomy.*

PATIENT	AGE	DATE OF SUBTOTAL GASTRECTOMY	DATE OF DIAGNOSIS OF TUBERCULOSIS
	yr.		
M.L.C.	60	5/12/50	Reactivated old "arrested" disease, 5/24/51
T.A.C.	32	2/14/50	Nov., 1952
G.H.H.	45	8/27/51	Dec., 1953
F.P.	61	1943	1952
R.F.S.	59	4/29/49	Oct., 1951
J.M.H.	33	1/10/44	Feb., 1952
T.L.C.	56	11/15/52	Reactivation of "arrested" disease, 8/4/53
L.E.W.	55	1945	Apr., 1947 (patient died in 1954 of cor pulmonale)
J.J.K.	43	Jan., 1948	Mar., 1948
H.J.B.	54	1/12/48	Jan., 1950
L.A.M.	52	Apr., 1950	Apr., 1953
J.P.	39	1945	Aug., 1952
H.D.	60	4/29/49	Reactivation from old focus of 1922 at about same time as hospitalization for subtotal gastric resection in 1950
R.T.	66	12/1/50	Reactivation of arrested disease found on admission for ulcer; patient died 14 days after subtotal gastric resection; tuberculous pneumonia found at autopsy.
L.E.F.	39	10/29/40	Mar., 1954
A.L.	59	10/10/51	12/25/51
W.H.B.	61	1947	Dec., 1952
P.J.D.	48	Mar., 1951	8/20/52
E.H.	40	1950	1953
J.A.D.	43	1949	1954
J.K.	54	Mar., 1948	Jan., 1949

SUMMARY AND CONCLUSIONS

Twenty-one patients with active pulmonary tuberculosis, 17 who had never been previously considered to have tuberculosis and 4 whose disease had been considered inactive, with reactivation after gastrectomy, are reviewed. Two patients died (a mortality of 9.5 per cent). The interval between gastrectomy and the diagnosis of active pulmonary tuberculosis averaged four years, but ranged between fourteen days and fourteen years.

Although the exact mechanism responsible for the development of tuberculosis after subtotal gastrectomy cannot be positively defined, it appears that the faulty assimilation of nourishment over a long period, together with metabolic imbalance brought about by chemical factors in the body, so lower a person's resistance that tuberculosis is a likely sequel in a certain proportion of patients. Since only 21 such cases have been collected in a 615-bed hospital with an annual admission rate of 700 in a period of more than five years, there does not appear to be cause for great alarm; however, when this operation is done one must reckon with the possibility of tuberculosis, and every means of preventing contact with an open case of tuberculosis should be stressed. X-ray examination of the chest should be made every six months for at least five years after operation. Furthermore, before a subtotal gastrectomy, each patient should be screened by x-ray study of the chest to rule out any pulmonary tuberculosis, either active or inactive, at the time and in cases with tuberculous lesions coverage by antituberculosis drugs might be advisable in patients selected for gastrectomy.

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CASE RECORDS
OF THE
MASSACHUSETTS GENERAL HOSPITAL

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CASE 41201

PRESENTATION OF CASE

A fifty-year-old man entered the hospital because of abdominal pain.

Two months previously, he was admitted to another hospital because of steady pain in the lower abdomen, which awakened him during the early morning hours. This pain occurred almost nightly, but very rarely during the day, and was relieved by sitting up. Six weeks before admission, the patient had begun to have severe, sharp midepigastric pains, which occasionally radiated to the interscapular and subscapular portions of the back and were unrelated to food. Two weeks later, a constant ache in the middle and lower dorsal portions of the back had begun after supper; it lasted a few hours and was not related to position, coughing, sneezing or activity. These pains continued intermittently and became more severe. Progressive constipation was also noted. Physical examination revealed abdominal tenderness on deep palpation 5 cm. below and to the left of the umbilicus and a firm, smooth prostate enlarged to twice the normal size. Routine laboratory tests, including tests for occult blood on two stool specimens, were negative. The blood nonprotein nitrogen, calcium, phosphorus and alkaline phosphatase determinations were all normal, as were a lumbar puncture and an electrocardiogram. An x-ray film of the chest showed bilateral apical pleural thickening. An intravenous pyelogram was normal. Films of the spine revealed a slight scoliosis to the right in the midthoracic region and small spurs in the lower lumbar vertebras. A barium-enema examination showed a "fuzziness" in the sigmoid, and an upper gastrointestinal series disclosed a tiny hiatus hernia present in the recumbent position and a minimal irregularity in the duodenal bulb. During the course in the other hospital the patient complained intermittently of pain in the lower back that was relieved by Empirin and of moderate abdominal pain after the evening meal, which often persisted into the late evening or early morning hours. At sigmoidoscopy a small sessile polyp 5 cm. above the anus was electrodesiccated. He was discharged on the ninth hospital day on a bland diet, Gelusil, tincture of belladonna and phenobarbital.

Despite this management, during the next five weeks, the pains repeatedly recurred and were relieved only by aspirin and codeine. He lost 10 pounds and noted mild anorexia, with occasional bloating and nausea on the bland diet. He had had no vomiting, hematemesis, melena, diarrhea, mucus in the stools, fever, chills or jaundice.

FIGURE 1. *Posteroanterior Film from Upper Gastrointestinal Examination, Showing Extrinsic Pressure on the Medial Aspect of the Duodenal Loop.*

There is constriction of the junction of the first and second portions of the duodenum, thought to be the result of a dilated common duct.

For about six years he had had intermittent episodes of vague abdominal distress, sometimes localized in the lower abdomen. During this period he had consulted a number of physicians but without any apparent specific diagnosis. A negative gastrointestinal "work-up" had been performed four years before admission.

Physical examination revealed a well developed, well nourished man, with slight tenderness to moderate pressure in the left midportion of the abdomen and hyperactive peristalsis. There was a questionable fullness in the epigastrium.

The temperature was 98.6°F., the pulse 80, and the respirations 20. The blood pressure was 120 systolic, 80 diastolic.

Urinalysis was negative. Examination of the blood showed a hemoglobin of 14.8 gm. per 100 cc. and a white-cell count of 11,100, with a normal differential count. On smear the red cells showed slight variation in size. The serum amylase was 13 Russell units per 100 cc. A blood Hinton test was negative. A finger stool specimen gave a + guaiac test.

On the second hospital day a prostigmine-morphine test was negative. The blood sedimentation rate was 22 mm. in one hour. The blood alkaline phosphatase was 4.8 units, and the bilirubin 0.3 mg. per 100 cc. A glucose tolerance test showed the following blood sugar levels: fasting 150 mg., half an hour after glucose 232 mg., one hour 268 mg. and two hours 325 mg. per 100 cc. A routine stool specimen was guaiac negative. On the fifth hospital day, an upper gastrointestinal series revealed a large phrenic ampulla, initial pylorospasm and a suspicious area of compression at the junction of the first and second portions of the duodenum possibly caused by a dilated common duct (Fig. 1). Sigmoidoscopy on the next day was negative except for intense spasm of the sigmoid and pain. A surgical consultant thought nodules were questionably palpable on the anterior aspect of the rectum. An attempted intravenous cholangiogram with 40 cc. of Cholografin revealed no opaque medium in the biliary passages an hour and a half after injection of the dye. Two additional stool specimens were guaiac negative. The sedimentation rate was 20 mm. in one hour. The prothrombin time was 70 per cent of normal. The bromsulfalein retention was 7 per cent in forty-five minutes. Two stool examinations showed normal neutral fat, soaps and other fat combinations, and failed to reveal any undigested muscle fibers. On the twelfth hospital day an operation was performed.

Differential Diagnosis

Dr. Greene Fitzhugh*: Dr. Wang, we might look at the x-ray films to see the constricted area.

Dr. C. C. Wang: In the gastrointestinal series the stomach is normal. The mucosal folds of the duodenal cap appear somewhat thickened, but I do not see any real deformity or ulcer crater; however, there appears to be a smooth extrinsic pressure defect on the upper end of the second portion of the duodenum medially, but actual ulceration is not seen in this region (Fig. 1).

Dr. Fitzhugh: Can you point out the dilated common bile duct?

Dr. Wang: I cannot confirm that but the duodenal loop is not widened. The spleen is not large. In the Cholografin test, there is no appreciable contrast medium in the common duct; no opaque stones are recognized.

Dr. Fitzhugh: In the gastrointestinal series, you found something that was not a diaphragmatic hernia; what was it?

Dr. Wang: The phrenic ampulla.

Dr. Fitzhugh: I am sure many of us do not know what a phrenic ampulla is; I found out from Dr. Stanley M. Wyman.

Dr. Wang: It is a normal dilatation of the lower end of the esophagus but no real herniation of gastric mucosa through the hiatus.

Dr. Fitzhugh: In summary this fifty-year-old man had pain in the lower abdomen to the left of the midline and an exploratory operation was done. The fuzzy sigmoid colon I thought I had better disregard in trying to arrive at a decision about why he had an exploratory operation. The ulcer regimen did no good. There were no consistent tests that led to the diagnosis of disease of the pancreas, but I have been led in that direction by the glucose tolerance curve and the pressure area demonstrated on the x-ray films. The symptom of pain radiating to the interscapular area and to the dorsal area of the spine that was unrelated to food was present during the other hospital admission. When I had read that far in the history, I thought that the story sounded like a retroperitoneal tumor. He was discharged from the other hospital on an ulcer regimen — bland diet, belladonna and so forth — but did not improve. I think that the physicians in the other hospital treated him for ulcer on the basis of the irregularity in the duodenum. They probably had not noticed the slight pressure defect in the first and second portions of the duodenum.

I shall briefly mention and rule out dissecting aneurysm of the aorta on the basis of the fact that the pain was not nearly severe enough; pain from that cause usually is severely agonizing and usually does not radiate down the arms. Moreover, at some stage, there is interference of blood supply to the head, to the extremities or to the kidneys. Other conditions that I do not believe he had are tuberculous or some other type of peritonitis, with extensive adhesions, which could have produced indigestion and constipation. I do not believe, however, that the pain would have radiated through to the back. Moreover, with obstruction, he would have been vomiting and would have had constipation. Meckel's diverticulum also would have caused the symptoms of obstruction.

The six years of a vague abdominal distress lead one to believe that he had a chronic pancreatitis or a chronic slowly progressing disease of the pancreas. A malignant process would not have lasted this long unless it was a giant-follicle lymphoma. I saw a man with giant-follicle lymphoma who returned for his second biopsy twenty years later still able to carry on his work. Thus, lymphoma can have a long history. I should not want to go so far as to make that diagnosis in this case. There was some fullness in the epi-

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gastrium but no definite masses. The glucose tolerance test showed a perfect diabetic curve and was consistent with disease of the pancreas. A diabetic curve results from any disease that destroys nine tenths of the pancreas. There was no history of sugar in the urine; the urine was reported as normal. The compression of the first and second portions of the duodenum, I believe, means only that he had disease of the pancreas.

Dr. William V. McDermott, Jr., who does the prostigmine-morphine tests, told me that the theory is that if morphine is given to a patient with pancreatic disease to constrict the common bile duct and then prostigmine is given, the prostigmine will inactivate the cholinesterase and allow the vagus nerve, a parasympathetic nerve, to build up more acetylcholine — in other words to stimulate the pancreas — and the blood amylase will rise. Many physicians think it is not a very practical test, but it is worth doing if the situation is puzzling. In the patient under discussion the amylase was normal so that it gives me no information.

Other diseases of the pancreas to consider are parasitic cysts, dermoid cysts and pseudocysts — cysts secondary to inflammation. They can be of any size. I think a simple cyst would not have disturbed the glucose tolerance curve. The chances are that it was not a cyst although I have no reason to say that except that the severity of the pain in the back and interscapular region suggests a retroperitoneal tumor.

The dilated common bile duct could have been a congenital disorder, but that is usually found in children. I do not remember seeing it in a patient of this age.

I believe that the patient was explored because the physicians did not know what he had and thought he might have had a carcinoma of the pancreas. Incidentally, he lost 10 pounds between the two admissions, and he had persistent discomfort. I think that he had a carcinoma of the pancreas although he may have had a cystic abnormality.

Dr. Benjamin Castleman: This man was a patient of Dr. Chester M. Jones and was operated on by Dr. Bartlett. Should you like to comment about the opinion on the ward before operation, Dr. Bartlett?

Dr. Marshall K. Bartlett: The line of reasoning through which we went was much the same as Dr. Fitzhugh has followed. The preoperative diagnosis was carcinoma of the pancreas. We believed that there was no way to make the diagnosis except to explore the patient.

Clinical Diagnosis

?Carcinoma of pancreas.

Dr. Greene Fitzhugh's Diagnosis

Carcinoma of pancreas.

Anatomical Diagnoses

Adenocarcinoma of pancreas, with peritoneal metastases.

Ascites.

Fatty vacuolization of liver, mild.

Pathological Discussion

Dr. Bartlett: When the abdomen was opened it contained about 300 cc. of cloudy, straw-colored fluid. There was extensive seeding with tumor of all the peritoneal surfaces, including the posterior cul-de-sac, which accounted for the peculiar masses that we had noted on rectal examination preoperatively. There was not a true rectal shelf but the beginning of such a process. The primary lesion was an extensive, stony hard, nodular tumor that involved the entire pancreas and also the gastrohepatic ligament. It was not possible to expose the common bile duct easily so we did not see that. The gall bladder was collapsed but otherwise not remarkable. We took a biopsy of the omentum, which was extensively infiltrated with tumor, and also a needle biopsy of the body of the pancreas. No further procedure was carried out.

Dr. Fitzhugh: I do not understand why, with a normal bromsulfalein retention of 7 per cent, no dye showed in the cholangiogram. The patient had good liver function. Do you know why, Dr. Wang?

Dr. Wang: No; I do not.

Dr. Castleman: This patient just two months previously had extensive physical and x-ray examinations but nothing on which to make a positive diagnosis was found. During that two-month period the tumor had grown rapidly so that it was easy with the long finger to feel the beginning of the rectal shelf. The histology indicated a very undifferentiated adenocarcinoma. It was spreading in perineurial lymphatics in the two biopsies. I gather that he is now receiving x-ray treatment for the back pain.

Dr. Bartlett: The pain is this man's outstanding problem at present. The Department of Radiology has agreed to give him a trial of therapy over this area of the back to see if it will relieve the pain.

The whole case is a good example of the difficulty we have in making the diagnosis of carcinoma of the pancreas. We have only once successfully operated on a patient with a tumor arising in the body of the pancreas; that patient lived only five months. We simply cannot make the diagnosis at a stage when complete surgical removal of the lesion is possible. I do not think I can tell what part of the pancreas contained the original focus in this case because tumor involved the entire gland by the time of exploration.

Dr. Wang: I have reviewed x-ray films taken in the other hospital two months previously. I do not believe the duodenal loop was adequately examined; otherwise, I believe the defect would have been seen then.

DR. ALFRED KRANES: Were any chest films taken in the last admission?

DR. WANG: The chest films were normal.

DR. CASTLEMAN: This is the type of case in which Dr. Kranes would expect to see lymphatic spread of metastases.

DR. KRANES: That is what I thought about.

DR. CASTLEMAN: I think that type of spread will develop before the patient dies.

CASE 41202

PRESENTATION OF CASE

A seventy-eight-year-old woman was admitted to the hospital because of difficulty in eating.

Five months before admission anorexia developed, and the patient found it "hard to eat." Two months later she had a vague ache in the upper abdomen, of several days' duration, which was not related to food intake and did not radiate. Two months before admission she began to gag and vomit with eating; she regurgitated "phlegm" and food, but no blood. She became so weak that she was virtually bedfast. At this point she was admitted to another hospital, where a barium-enema examination, upper gastrointestinal series, and gall-bladder study were reported negative. The hemoglobin was 10.3 gm. per 100 cc., and the white-cell count 14,000, with a normal differential. The blood nonprotein nitrogen was 34 mg., and the fasting blood sugar 124 mg. per 100 cc. Treatment with chlorpromazine (Thorazine), tincture of belladonna and digitoxin resulted in temporary improvement. However, the symptoms soon returned, and the diet was confined almost entirely to fluids. On the day before admission, after swallowing, she felt "something sticking" in the midportion of the chest. The total weight loss at the time of admission was 25 pounds.

FIGURE 1. *Anteroposterior View of the Lower Left Side of the Chest with Barium in the Esophagus and Stomach, Showing Distal Esophageal Narrowing and Collapse of the Basilar Segments of the Left Lower Lobe.*

FIGURE 2. *Spot Film from the Gastrointestinal Examination, Showing Narrowing of the Esophagus and Extrinsic Pressure on the Left Aspect of the Lower Esophagus and the Medial Superior Aspect of the Fundus.*

She reputedly had had a "gall-bladder attack" with local tenderness and transient jaundice several months before the onset of the present illness. The family and social histories were noncontributory. She admitted a cough, productive of small amounts of white sputum. For three months she had noticed some shortness of breath and had had mild orthopnea. There was no history of hematemesis, melena, fever, chills, fatty food intolerance or frothy or foul stools.

Physical examination revealed a pale, deaf woman who appeared chronically ill. There were thickened

tympanic membranes, bilateral cataracts and ready elicitation of the gag reflex. The anteroposterior diameter of the chest was considerably increased, and respiratory expansion was decreased. The chest was clear to percussion and auscultation. A marked dorsal kyphosis was observed. The heart was thought to extend to the midclavicular line in the sixth interspace, and a Grade 1 to 2 apical systolic murmur was heard. The liver edge was palpable 1 fingerbreadth below the right costal margin. Bilateral axillary adenopathy was noted. Deep tendon reflexes were hyperactive.

The temperature was 99.4°F., the pulse 120, and the respirations 24. The blood pressure was 132 systolic, 94 diastolic.

The urine was normal. Examination of the blood revealed a hemoglobin of 10.4 gm. per 100 cc. and a white-cell count of 15,600, with a normal differential. The nonprotein nitrogen was 35 mg., the fasting blood sugar 173 mg., the amylase 8 Russell units, the alkaline phosphatase 58.8 units, the bilirubin 0.8 mg., the total protein 5.8 gm., the albumin 3.2 gm., and the globulin 2.6 gm. per 100 cc. A stool guaiac test was negative, as was a blood Hinton test. An x-ray film of the chest showed some cardiac enlargement in the region of the left ventricle and a very tortuous aorta (Fig. 1). The left leaf of the diaphragm was not clearly seen, and there appeared to be considerable increase in the bronchovascular markings in this area. Generalized pulmonary emphysema was noted. A barium-enema examination was negative. An upper gastrointestinal series on the sixth day showed a narrowing of the distal esophagus, with delay in the passage of barium interpreted as being most likely due to extrinsic pressure but possibly due to a neuromuscular disturbance or esophagitis (Fig. 2). X-ray films of the bones revealed considerable demineralization, but no metastases. On the fifteenth hospital day the serum cholesterol was 151 mg., and the bilirubin 0.3 mg. per 100 cc., and bromsulfalein retention was 15 per cent. Thymol turbidity was 2.0 units, and cephalin flocculation was negative in forty-eight hours. The prothrombin time was 17 seconds (38 per cent of normal).

Esophagoscopy revealed a 2-cm.-deep erythematous band encircling the entire esophagus at a point 13 cm. above the cardia. This was interpreted as esophagitis with narrowing of the esophagus. No ulcer was seen below this area. Biopsy specimens revealed no diagnostic abnormality. Therapy now included menadione bisulfite (Hykinone) parenterally and, just before meals, nitroglycerin sublingually. Three days later the prothrombin time was normal. A string was passed down the esophagus. On the twenty-sixth hospital day, peritoneoscopy revealed a small slightly granular liver. A liver biopsy showed severe fatty vacuolization. A bone marrow aspirate demonstrated no diagnostic abnormality. Another esophagoscopy revealed a large amount of food in the proximal esophagus and less evidence of frank esophagitis. Dilatation up to a size No. 22 bougie was carried out. The swallowed string was found in the esophagus. A No. 24 bougie could not be passed. Ten days later an unsuccessful attempt was made to pass a gastric feeding tube, and at a third esophagoscopy bougienage was unsatisfactory. Three biopsies were reported as showing slight chronic inflammation. Two days later an electrocardiogram showed frequent atrial and ventricular ectopic beats, hypertrophy of the left ventricle and nonspecific T-wave changes consistent with coronary-artery disease.

On the forty-fourth hospital day a Stamm gastrostomy was accomplished. On the second postoperative day the patient was given skim milk and vitamin supplements by the gastrostomy; she vomited part of two feedings. The serum sodium, which had been 132 milliequiv. per liter, had dropped to 121, and the chloride from 90 to 80 milliequiv. per liter. On the ninth postoperative day leakage around the gastrostomy first appeared. Three days later a Foley catheter was placed in the stomach, and gentle traction was applied to prevent the gastrostomy leakage. The patient was disoriented. The cardiac rate was 120, with an irregular rhythm. Sodium-free fluids were given intravenously and by gastric tube. Digitalization was carried out, and chloramphenicol (Chloromycetin) therapy was begun. The serum sodium was 160 milliequiv., and the chloride 128 milliequiv. per liter, and the nonprotein nitrogen 73 mg. per 100 cc. An electrocardiogram showed a shift in the axis to the right and inverted T waves in the precordial leads, suggesting the possibility of a pulmonary infarct. The tracing also revealed atrial premature beats and hypertrophy of the left ventricle. On the thirteenth postoperative day the serum sodium was 131 milliequiv., and the chloride 94 milliequiv. per liter, and the nonprotein nitrogen 25 mg. per 100 cc. By the sixteenth postoperative day diarrhea had developed. The patient was still losing much of the feedings around the gastrostomy tube, which was replaced with a larger tube. On the next day the serum nonprotein nitrogen was 46 mg. per 100 cc., and the sodium 140 milliequiv., the potassium 2.3 milliequiv., the chloride 108 milliequiv., and the carbon dioxide 18 milliequiv. per liter. The output of urine was low and diarrhea persisted. The patient became semicomatose and died two days later.

Differential Diagnosis

Dr. Earle W. Wilkins, Jr.*: This is the account of an elderly woman, with a nonspecific chief complaint. The protocol illustrates the difficulties that faced her physicians in the establishment of a single diagnosis. I have the advantage of the entire story but the disadvantage of not seeing the patient.

The history of anorexia, vague pain in the upper abdomen, difficulty in eating, vomiting, weakness, weight

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loss and anemia suggests a malignant process; the exact location of the process is not clear. Coincidental with admission, or actually one day previously, the first localizing symptom, dysphagia, developed. Much of the hospital course was devoted to the apparently unsuccessful attempt to elucidate the nature of this complaint. During this investigation a group of additionally confusing findings was forthcoming. These included a markedly elevated serum alkaline phosphatase, an abnormal bromsulfalein retention, a prolonged prothrombin time, a small granular liver by peritoneoscopy and severe fatty vacuolization by liver biopsy. The common denominator of these findings is liver disease. The final portion of the protocol deals with some of the vagaries in the postoperative management of the elderly patient.

My problem, therefore, is to tie together, preferably in a single diagnosis, a history compatible with a malignant process, a cause for dysphagia and liver disease of unknown type. These, along with an unsuccessful gastrostomy, resulted in the death of the patient. I shall attempt a diagnosis by focusing primarily on the dysphagia.

The two most important points in the diagnosis of the cause of dysphagia are the history and the roentgenologic examination. The physical examination is not often helpful. Esophagoscopy is a useful adjunct and is best utilized when the findings resulting therefrom may alter the course of management. Cytologic study, although not mentioned in this case, is often helpful. Other methods of study of the esophagus — balloon studies, intraluminal-pressure recordings and secretion analyses — have no bearing in this patient.

May I see the x-ray films?

DR. C. C. WANG: The films of the chest (Fig. 1) taken on admission show an area of increased density in the medial basal segment of the left lower lobe, which is slightly reduced in size. There is no definite soft-tissue mass in the left hilar region. No enlarged lymph nodes are evident. The remaining lung fields are clear. The pulmonary vessels are somewhat congested. The heart is enlarged, without characteristic configuration, and the aorta is quite tortuous. The costophrenic sulci are clear. No evidence of metastasis is apparent in the bony thorax. A marked dorsal kyphosis is again seen. A gastrointestinal examination reveals that the esophagus is at the upper limit of normal in width. There is apparently a smooth extrinsic pressure defect at the lower end of the esophagus on the left, best seen on the spot film (Fig. 2). This asymmetrical pressure defect appears to extend continuously for a considerable distance to the medial aspect of the gastric fundus. The lowermost portion of the esophagus is markedly narrowed, but the mucosal folds give no evidence of ulceration. The barium-filled distal stomach shows no intrinsic defects. The gastric fundus, however, does not appear to be fully distended, but I cannot visualize any definite ulceration or tumor mass in this area. The remainder of the gastrointestinal tract is not remarkable.

DR. WILKINS: The prime causes of dysphagia are carcinoma of the esophagus or cardia of the stomach, peptic or corrosive esophagitis, achalasia, benign tumors, hiatus hernia and extrinsic pressure. Congenital defects — stenosis, webs and the "lower esophageal ring" of Schatzki and Gary[1] — are not likely at seventy-eight years of age. Less common causes include diverticulum, scleroderma and certain neurologic diseases such as myasthenia gravis and localized cerebrovascular accidents, for which there was no evidence. Foreign bodies usually cause dysphagia only when compounded on an underlying narrowing from another cause.

The history is not that of carcinoma of the esophagus. In this form of neoplastic disease dysphagia usually precedes the other symptoms that were mentioned in this case. The dysphagia is progressive, resulting in weight loss before the systemic effects of the malignant process become manifest. Carcinoma of the esophagus does not involve the liver until late in its metastatic course. Its early metastases are to the local lymph nodes, usually in descending fashion. The venous drainage of the esophagus is to the systemic circuit, thus sparing the portal bed until late. Finally, these x-ray films do not suggest carcinoma of the esophagus. Failure to find esophageal carcinoma by the esophagoscope does not exclude its presence.

Carcinoma of the cardiac end of the stomach could have produced all the symptoms presented by this patient before admission and the development of dysphagia. The dysphagia could then have resulted from extension to involve the esophagus directly or from involvement of the paracardial group of lymph nodes. Carcinoma of the stomach commonly involves the liver, often early, and could explain the liver disease. Recently, Lemon et al.[2] stated: "In patients without bone disease, elevation of the serum alkaline phosphatase is frequently one of the few reliable indications of hepatic metastases, even in the absence of other signs of biliary obstruction." One would have suspected, although it is not necessarily so, that either peritoneoscopy or the liver biopsy would have suggested metastatic disease in this case. The x-ray findings are not typical of carcinoma of the stomach, but the detection of cancer in the region of the cardia is notoriously difficult. The percentage of incorrect diagnoses is greater here than in any other portion of the gastrointestinal tract. There is the suggestion of an extrinsic mass adjacent to the cardia and compressing the distal esophagus. This is consistent with carcinoma, particularly with lymph-node involvement.

Lymphomatous lymph nodes and carcinoma of the body of the pancreas with nodal metastases likewise could produce this picture but are statistically less likely.

The strongest evidence for esophagitis lies in the gross observation at the first esophagoscopy, when an

erythematous band was visualized encircling the esophagus, and in the biopsy report of chronic inflammation from the third esophagoscopic examination. The band was seen 13 cm. above the cardia, which does not coincide with the interpretation of the x-ray films of the location of the point of narrowing. The findings of esophagoscopy and biopsy were not consistent, however, with the typical findings in peptic esophagitis. No erosions and no ulcerations were seen. The history was not that of peptic esophagitis in the absence of a previous history of dyspeptic symptoms, regurgitation or actual peptic ulcer. There was no history of ingested corrosives. Finally, the x-ray films are not typical of esophagitis unless merely in the hyperemic early stenotic phase without stricture or ulceration. The symptoms were of a more advanced obstruction.

Achalasia can be promptly dismissed. Although it has been reported with an onset in the eighth decade of life neither the x-ray pattern nor the esophagoscopic findings substantiate such a diagnosis. Measurement of intraluminal esophageal pressures in response to intravenous injection of methacholine (Mecholyl) has been reported[3] as diagnostic of achalasia if real doubt exists in a given case.

Benign tumors of the esophagus, the so-called intramural, extramucosal spindle-cell tumors, present a characteristic x-ray appearance of a smooth bulge into the barium contour. This appearance was not observed in the case under discussion. Such tumors are actually quite uncommon. Only 23 have been reported from surgical and autopsy specimens from this hospital.[4] A leiomyoma or other intramural gastric tumor could theoretically present this picture by a large extramural extension, but the chronic illness, the liver pattern and the esophagoscopic findings are not explained by such a diagnosis.

There was no history and no x-ray evidence of hiatus hernia.

At this level of the esophagus extrinsic pressure means involvement of esophageal and paracardial lymph nodes by carcinoma, lymphoma or, less commonly, tuberculosis or other granulomatous lesion. There was no evidence for these inflammatory diseases. A metastatic malignant process of the liver can cause obstruction of the lower esophagus by extrinsic pressure or direct invasion from without. A tortuous descending thoracic aorta, especially when accompanied by an enlarged heart, has been said to offer distal esophageal obstruction, although never so marked as in this case.

Scleroderma is in many ways an intriguing possibility. The patient's symptoms are consistent. The gastrointestinal series suggested a neuromuscular disturbance. The esophagoscopic findings are not incompatible. One cannot explain, however, an apparent mass outside the esophagus and liver impairment. I am not familiar with a case in which scleroderma of the esophagus was the only manifestation of the disease.

There are several additional factors in which I should be interested. Although she was in the hospital sixty-three days, no x-ray examination of the esophagus was made after the sixth day. I should like to have seen what subsequent films of the esophagus showed. I should like to have known what the stomach looked like at the time of gastrostomy, whether the palpation of the hiatus and paracardial group of lymph nodes was normal, and what the pancreas and liver looked like. The behavior of the gastrostomy raises the possibility of pyloric or duodenal obstruction, possibly by the pancreas or enlarged lymph nodes.

I cannot sufficiently explain the entire picture by liver disease alone. I have the choice of making the diagnosis of a fatty liver together with some condition such as esophagitis or scleroderma, or a single diagnosis of a malignant process. I shall choose carcinoma and suggest that it arose in the region of the cardia of the stomach, with submucosal extension into the esophagus, and with paracardial-lymph-node and hepatic metastases.

DR. AUSTIN L. VICKERY: Dr. Benedict, will you tell us what you saw at esophagoscopy?

DR. EDWARD B. BENEDICT: At the first two esophagoscopies, which Dr. Roger Wilcox and I did together, we were reasonably satisfied that the gross appearance of erythema and narrowing was due to esophagitis and benign stenosis. The biopsy report, however, showed no diagnostic abnormality. Negative biopsies, since they may not penetrate deeply enough or may not be taken from exactly the right spot, cannot be relied upon to exclude inflammatory or malignant processes. On the third esophagoscopy, only ten days later, there was, at a point 12 cm. above the cardia, a very firm, red, smooth, annular lesion, which was so hard on forceps palpation that I felt sure it must be malignant. Several large satisfactory biopsies were taken with cutting forceps, but the pathologist reported only slight chronic inflammation, with no evidence of a malignant lesion. In spite of this report we believed that we were dealing with carcinoma — most probably primary in the esophagus.

CLINICAL DIAGNOSES

Esophageal stricture, ?carcinoma.
Fatty infiltration of liver.

DR. EARLE W. WILKINS, JR.'S, DIAGNOSIS

Carcinoma of cardia of stomach involving esophagus, periesophageal lymph nodes and liver.

ANATOMICAL DIAGNOSIS

Bronchogenic carcinoma, left lower lobe, with invasion of wall of esophagus, pleura, pericardium, heart, diaphragm, peritoneum, liver and mediastinum.

Pathological Discussion

Dr. Vickery: At autopsy a large tumor mass was found involving the lower lobe of the left lung, the mediastinum, the superior aspect of the right lobe of the liver, the pericardium and the myocardium. The

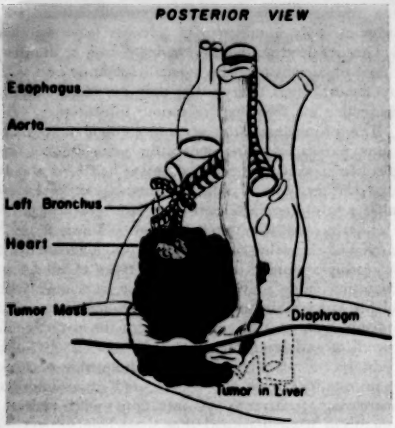

Figure 3.

bronchial wall of the posterior basal segment of the left lower lobe was roughened, hard and contiguous with the infiltrating neoplasm. The tumor proved to be a poorly differentiated squamous-cell carcinoma, with areas of central necrosis, and was considered to be bronchogenic in origin. The aggressive growth pattern of this carcinoma was evidenced by its penetration through the diaphragm into the liver and similarly through the pericardium into the wall of the left ventricle for a distance of 1 cm. The esophagus was distorted in its lower segment by the mass, being deviated to the right, near the gastric cardia (Fig. 3). There was only superficial invasion of the external muscular coat of the esophageal wall by the tumor, accounting for the repeated negative esophagoscopic biopsies at the area of constriction.

The liver showed a moderate degree of fatty change, which may have been related to the patient's prolonged poor nutritional status.

The clinical signs of cardiac irritability with premature beats, in retrospect, were possibly associated with the myocardial invasion by tumor. In addition there were small coronary-artery emboli, traceable to a mural thrombus in the left atrial appendage, which could also have contributed to the picture.

Dr. Benedict: Metastatic tumors involving the esophagus are rare but should be kept in mind.[5] In 1944 Toreson[6] reviewed 3048 consecutive autopsies in adults and found 599 cases of cancer, in 19 of which there was secondary involvement of the esophagus, a rate of 3.2 per cent. He also found 7 cases of secondary tumor of the esophagus, making a total of 26 cases in all. Of these, 24 were carcinomas, 1 was a lymphosarcoma, and 1 was probably a melanoma. The organs in which the tumors originated and the numbers for each were as follows: trachea 8, stomach 7, larynx 4, breast 2, pancreas 1, testes 1, metastatic lymph nodes 1, and origin undetermined 1. Most of the cases considered to be metastatic tumors are the result of direct invasion from neighboring organs or from mediastinal lymph nodes. However, in 2 of the breast cases described by Toreson,[6] a metastatic tumor nodule was found in the wall of the esophagus without involvement of the mediastinal lymph nodes.

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ANTIBIOTICS AS PROPHYLACTIC AGENTS

MANY practicing physicians, if not the majority, use antibiotics for purposes besides the treatment of active infection. For the most part these uses are for conditions in which serious infections supervene and are an important cause of death. Unfortunately, the practice has become so widespread that in most hospitals only a minority of patients leave without having received antibiotics for one reason or another. Some physicians give the agents reluctantly and with apologies; others believe that they are really helping to reduce the frequency of serious infections.

The prophylactic use of antimicrobial agents became particularly prevalent after penicillin became plentiful and its cost reduced and it had been shown that certain types of infection, notably streptococcal pharyngitis, puerperal sepsis, and later gonococcal infections, could thus be prevented. However, in a survey of the value of drugs used prophylactically, it is readily apparent that the only types of infections that they have prevented are those associated with highly susceptible organisms that, when they cause infection, are readily controlled with the same agents given for early and intensive treatment.

In recent years the increasing frequency of toxic manifestations from the widely used drugs and the increasing occurrence of infections with resistant organisms have suggested that the whole subject requires reappraisal. Proper and adequate data are difficult to obtain and to interpret, but an increasing number of secondary infections or superinfections are reported in patients being treated with antibiotics, even for specific infectious diseases with susceptible organisms. Nevertheless, these recent developments do not seem to have deterred most physicians from continuing with the widespread administration of antibiotics in prophylaxis.

One situation in which the prophylactic use of antibiotics is probably universal and is considered to be justified is when tracheotomy has been performed on patients with poliomyelitis who have difficulty in breathing, often to the point where a respirator is required. In such patients who recover from the acute stage of the disease, death frequently occurs as the result of pneumonia. This is caused by the failure of the patient to rid the trachea of secretions, permitting the infection to invade the atelectatic areas of the lung. Secretions from these patients offer a good opportunity for investigation of the prophylactic uses of antibiotics.

At the Municipal Contagious Disease Hospital in Chicago, Lepper and his associates* have recently conducted such a study in 72 tracheotomized patients. The bacterial flora of the tracheal secretions was determined before and at stated intervals after the tra-

*Lepper, M. H., Kolman, S., Blatt, N., Dowling, H. F., and Jackson, G. G. Effect of eight antibiotics used singly and in combination on tracheal flora following tracheotomy in poliomyelitis. *Antibiotics & Chemother.* 4:829-843, 1954.

cheotomy. All the organisms recovered were considered potentially pathogenic because similar organisms were recovered from the blood and organs at autopsy. The authors therefore observed the effects of various antibiotics, singly and in combination, on the retention or clearing of the flora initially found and on the implantation of new organisms on the tracheal flora (which was compared with the findings when therapy was withheld). A total of 391 intervals were studied, during 137 of which no antimicrobial therapy had been given. The results of the study are indeed illuminating.

In the first place, pneumococci, Group A hemolytic streptococci and *Haemophilus influenzae*, which were the most common causes of fatal terminal pneumonias before the introduction of chemotherapy, were not found in any of the patients — either during periods when they were receiving treatment or in the intervals between treatment. Whenever these organisms were found they were readily eliminated, and treatment of infection caused by them has been most successful. Infections with these organisms may therefore no longer be considered an important indication for prophylaxis in any case in which it is possible to follow the patient both clinically and bacteriologically and to institute treatment promptly.

The effect of antibiotics on other organisms depended on the types that were found. Against gram-positive coccal infections — namely, enterococci, *Streptococcus viridans* and *Staphylococcus aureus* — none of the antibiotics but polymyxin* and against the staphylococcus no agents but the sulfonamides favored the implantation of these organisms; in other words, these were no more frequent in treated than in untreated patients. On the other hand, the antibiotics besides polymyxin may have reduced the frequency with which these organisms become implanted, although a combination of two or more agents other than polymyxin or the sulfonamides was necessary to implant *Staph. aureus*. Combinations of antibiotics favored the removal of enterococci and *Str. viridans*, but the use of single antibiotics may actually have facilitated the retention of enterococci. Whereas none of the antibiotics favored the removal or retention of the staphylococci, combinations of antibiotics helped the implantation of new strains.

There were some differences among strains of gram-negative bacilli, but actually only polymyxin prevented or reduced the frequency of implantation of pseudomonas and some of the broad-spectrum antibiotics lessened the chances of implanting aerobacter. Although these agents did not facilitate the retention of aerobacter, the use of broad-spectrum antibiotics did favor the retention of both pseudomonas and proteus organisms. None of the antibiotics prevented the implantation of candida (monilia), but any sort of treatment with any antimicrobials or combination of antibiotics favored the implantation of these organisms and their retention.

The authors concluded that, regardless of the number and type of antibacterial agents used, the prevention of implantation of one or more organisms into a sterile trachea is only slightly more effective than no treatment at all. Moreover, no combination or single agent increased the frequency of bacteriologic improvement over that occurring spontaneously. In fact, the broad-spectrum antibiotics greatly reduced the prevalence of the favorable change in the tracheal flora. All combinations and single agents, except when three or more drugs were used, increased the number of organisms in the trachea more than that which occurred with no treatment, and this was particularly noticeable when the so-called broad-spectrum agents were given. The combination of broad-spectrum and limited-spectrum agents was more likely to maintain the status quo than no treatment and less apt to cause replacement of one species by another. The authors are of the opinion that, in spite of the possibility of preventing pneumococcal, streptococcal and *H. influenzae* infections, the unfavorable alteration of flora that occurs when prophylaxis is attempted probably outweighs the good that may be accomplished. Therefore, it is preferable to follow the bacteriologic aspect closely and use a specific agent only when the clinical condition warrants it.

If this conclusion is reached after such a study, is it not important to reassess the prophylactic administration of antibiotics in all situations except when they have definitely proved of value and then only for highly specific purposes?

*Polymyxin B was given by aerosol; all other antibiotics were given systemically.

POLIOMYELITIS VACCINE

THE editors of the *Journal*, awed by the incandescent publicity accompanying the revelation that a vaccine of practical effectiveness against poliomyelitis may be at hand, have so far refrained from making any statement. Since any word of caution against a too ready acceptance of the thesis that poliomyelitis will shortly become a disease of the past would have been insignificant, unpopular and unheeded, they have successfully controlled any urge to contribute to the shouting and the tumult.

The difficulties of producing a vaccine effective against a virus infection are too well known in principle and too complicated in practice to be gone into here. They include the possibility that a killed virus may be ineffective and that an attenuated one may be dangerous, that a practical test for immunity is lacking and that no one has reliable knowledge concerning the duration of protection and the matter of possible undesirable sensitizations — all these are problems with which the experts are constantly laboring.

When the fact is taken into account that the present supply of vaccine seems also quite inadequate to meet the demand that has been created, it becomes

apparent that the situation requires a calm appraisal. Such an appraisal has been made for this state by the Massachusetts Poliomyelitis Vaccine Committee, serving as an advisory body to the Commissioner of Public Health, and a plan for priorities when the distribution of the vaccine is sanctioned has been formulated. This plan has been endorsed by the Executive Committee of the Massachusetts Medical Society, meeting in extraordinary session, and the services and whole-hearted co-operation of the Society are assured.

There seems to be, on the whole, little occasion for permitting recent advances in the steady attack on infectious disease, however spectacular, to become the basis for an epidemic of major mass hysteria.

DISCOURSE AND LECTURE

THE current issue of the *Journal*, without curtailing in any way the more distant interests that it serves, can truly be considered as a Massachusetts Medical Society number. Not only has it made its appearance during the week of the Society's hundred and seventy-fourth annual meeting; it contains also, perhaps for the first time, both the annual discourse and the Shattuck Lecture in the same timely edition.

These two articles, of general interest wherever they may be read, exemplify perfectly the patterns into which custom has decreed that they should fall. Dr. Bearse's exposition of the plight of the voluntary hospital, a social and economic problem, follows the philosophical trend established for these essays early in the nineteenth century.

Dr. Child's Shattuck Lecture on the portal circulation, with special reference to portal decompression by surgery, presents the results of his experience at the New York and New England Center hospitals. It stands as a worthy addition to the series of clinical and scientific papers produced under this lectureship since the first of the line was delivered in 1890 by Dr. George B. Shattuck, grandson of the donor, on the subject "Influenza in Massachusetts."

STRAIGHT THINKING AND SIMPLE JUSTICE

THE announcement by Dr. Curtis C. Tripp that payments to physicians for in-hospital care of welfare recipients will be resumed, coming at this time, is the crowning achievement of his administration as president of the Massachusetts Medical Society. It represents, not a victory for the physicians over the Department of Public Welfare, but the establishment of a clear principle of straight thinking and simple justice. As neatly expressed by the councilors of Middlesex South district:

> Poverty of a patient commands the gratuitous services of a physician. When, however, the cost of medical service to the poor, together with that of other necessities of life, is assumed by local, state or federal government agencies by means of tax money of which an equitable share has been paid by physicians, there is no obligation for him to contribute further of his time, skill or resources unless for the benefit of the poor patient in a teaching hospital or one who is not eligible for public relief.

It also demonstrates that quiet, persistent and unruffled personal discussion may be a better method of establishing such a principle than vociferous demands (however well intentioned) that "there ought to be a law" compelling the State to pay doctors for everything they do for the recipients of welfare.

Lupulin as an Anaphrodisiac. — *Lupulin, or the active principle of hops, is possessed of powerful sedative effects on the generative functions . . .*
Boston M. & S. J., *May 24, 1855*

MASSACHUSETTS MEDICAL SOCIETY

ARTHUR ALDEN CUSHING
1881-1955

For the greater part of the past fifty years two doctors named Cushing lived in Brookline. Sharing a common ancestor six generations back, they had pioneer grandfathers in the Midwest, and each eventually settled in his native New England to practice medicine. One became world famous, though little known to the common folk of Brookline. The other, Arthur Alden Cushing, won high regard among a privileged group of medical associates — and almost abject devotion from a succession of grateful patients.

Alert to changes in modern medicine, anxious ever to improve his knowledge and ability, Arthur Cushing made his greatest contribution in conscientious observation and reobservation — coupled with kindness, sympathetic understanding and an unlimited capacity genuinely to "care" for his patients. His Puritan background and consecrated resolve to do what he considered "right" for his patients and in all aspects of life occasionally drove him to a course alien to his normal sweetness of disposition. But no one could question his sincerity, and few failed finally to understand.

A perfectionist, conscientious to a fault, giving untold hours of selfless labor to whatever cause needed his help, he nevertheless preferred to remain in relative obscurity and sought no fame. Although Arthur Alden Cushing became more and more a power and almost a tradition in his community, he himself would wish to be remembered (in this modern era of

"bed-end medical practice") as an old fashioned family doctor and friend confessor of his patients.

F. W. M.

COMMITTEE ON MATERNAL WELFARE

Obstructing Carcinoma of the Cervix

A thirty-six-year-old gravida VI, para V, was admitted to the hospital in active labor a month postmature according to dates. She had seen a physician only twice during pregnancy and had not had a pelvic examination. Bleeding was denied. She made no progress in twenty-four hours, and a consultant was called. He made a diagnosis of a fungating obstructing carcinoma of the cervix and delivered a live baby by Kerr cesarean section, biopsying the cervix from below immediately after operation. The course was uncomplicated until the third day, when distention developed with a temperature of 104°F. and a pulse of 160. Intubation, transfusion and antibiotics (for presumed peritonitis) were administered, but the patient went into circulatory collapse, responding for a while to appropriate measures but suddenly deteriorating again, and she died on the fourth postoperative day. Autopsy confirmed extensive cancer but gave no evidence of peritonitis, and a blood culture was negative after sixteen days.

This case was not classified for preventability, because the immediate cause of death was not known. There was unanimous agreement, however, that the prenatal care had been inadequate, and that one thorough pelvic examination, which should have been performed early in pregnancy, would have revealed the carcinoma. These factors, however, did not contribute directly to the patient's death. Twenty-four hours of active labor without progress was too long a delay before the consultant was called, particularly in a gravida VI, para V, who, under other circumstances, might well have come to grief during this time from obstructed labor. Again, it is not implied that this delay caused the patient's death. The management from that point on seemed unusually good; nevertheless, even though she was suffering from a probably incurable disease, the postoperative death was untimely.

John Figgis Jewett, M.D.
Chairman

DIRECTORY OF FELLOWS

A new *Directory* of the officers and fellows of the Society has been published, corrected to January 1, 1955.

Copies are available to all fellows who apply to the Secretary, 22 Fenway, Boston 15.

DEATH

Sostek — Samuel B. Sostek, M.D., of Malden, died on May 1. He was in his forty-sixth year.

Dr. Sostek received his degree from Boston University School of Medicine in 1937.

He is survived by his widow, parents, a daughter, a son and three brothers.

BOSTON MEDICAL LIBRARY

BIOGRAPHY

The third volume of *La galerie des hommes célèbres* issued by Lucien Mazenod of Geneva is devoted to celebrated physicians.* It was published under the direction of Drs. René Dumesnil and Flavien Bonnet-Roy. The text, which is illustrated with full-page portraits, is divided into epochs, beginning with Imhotep of Egypt (2980 B.C.) and ending with Harvey Cushing (nineteenth and twentieth centuries). Appended to the text is a valuable list of 59 pages of eminent physicians beginning with mythologic times, Greece and Rome and physician saints to historians and littérateurs of the nineteenth century.

**Les médecins célèbres.* 371 pp. Geneva: Lucien Mazenod, 1947.

MEDICAL PAMPHLETS

The Library has received an interesting volume of pamphlets from Dr. William B. Bartlett. Among other items it contains William Douglas's *History of a new epidemical eruptive miliary fever, with an angina ulcuscula which prevailed in Boston New England in the years 1735 and 1736.* (Boston: 1736). Another pamphlet is the dissertation (one of the earliest Harvard medical dissertations to be printed) of Peter de Sales la Terriere on puerperal fever, delivered for the degree of Bachelor in Medicine at the University of Cambridge in America (Harvard) in 1789. It is a presentation copy from the author inscribed to Dr. Josiah Bartlett, who was prominent in the affairs of the Massachusetts Medical Society and active in its reorganization in 1803 and its president in 1862-1864.

John Fleet was one of the two first graduates of Harvard Medical School in 1788, the other being William Pearson and the third being Peter de Sales la Terriere. John Fleet was the first librarian of the first Boston Medical Library organized in 1805. This was the second proprietors' library in Boston, preceded by the Social Law Library, still in active existence.

CORRESPONDENCE

HEART DISEASE AND WORKMEN'S COMPENSATION

To the Editor: I welcomed the special article, "Heart Disease in Massachusetts in Relation to the Workmen's Compensation Act," by Clark, Tousant and Sprague, as well as the editorial comment entitled "Economic Dilemma," in the March 24 issue of the *Journal.*

A documented study of 100 cases of workmen's compensation pointing out these recurrent problems of heart disease and industry was partially underwritten by the New York Heart Association and recently published (Meyer Texon, *Heart Disease and Industry with Particular Reference to Workmen's Compensation Cases*, New York: Grune and Stratton, 1954).

The experience in Massachusetts seems to be almost identical with ours in New York State. It is likely that the remaining states in the Union have had a similar experience.

It should be emphasized that one must not confuse the requisites of scientific proof in achieving the beneficent purposes of workmen's compensation laws. The scientific facts concerning the question of causal relation with reference to problems of atherosclerotic heart disease usually weigh against the worker. Yet social evolution frequently favors the worker through a sympathetic administration of the law. If benefits are to be broadened the desirable course lies in a law providing comprehensive benefits for disability from accident or illness. Thus, all questions of fault, time, place and causal relation could be eliminated. Law, science, industry and society would all be served realistically.

MEYER TEXON, M.D.
New York City

BOOK REVIEW

Clinical Management of Behavior Disorders in Children. By Harry Bakwin, M.D., professor of clinical pediatrics, New York University, visiting physician, Bellevue Hospital, and attending pediatrician, University Hospital; and Ruth M. Bakwin, M.D., associate professor of clinical pediatrics, New York University, associate visiting physician, Bellevue Hospital, and director of pediatrics, New York Infirmary. 8°, cloth, 495 pp., with 14 illustrations. Philadelphia: W. B. Saunders Company, 1953. $10.00.

As the years pass, increasing attention is being given to behavior disorders in children. This new book on the clinical management of such conditions is remarkably thorough in its arrangement and expression. Topic by topic, it takes up and in a clear and practical way describes and discusses the main problems parents and teachers encounter.

This volume is a fine example of the improvement in format and arrangement in the publication of medical books in the United States; in about 500 pages, the authors have condensed just about everything important on the subject that has appeared in the last fifty to seventy-five years in this country and abroad. The work is slanted directly toward treatment that will make it especially useful to general practitioners as well as pediatricians. Some psychiatrists may already be familiar with much of the material, but the preparation by this team of authors has been thorough; they have brought the contents completely forward and have expanded some earlier ideas by correlating them with the most recent work.

MERRILL MOORE, M.D.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

The Psychiatric Interview. By Harry Stack Sullivan, M.D. Edited by Helen Swick Perry and Mary Ladd Gawel, with an introduction by Otto Allen Will, M.D. 8°, cloth, 246 pp. New York: W. W. Norton and Company, Incorporated, 1954. $4.50.

The text of this book is based on two lectures that the late Dr. Sullivan gave in 1944 and 1945 under the auspices of the William Alanson White Psychiatric Foundation. They present the technic of the personal inquiry.

Cerebrovascular Disease. By James Peter Murphy, M.D., assistant clinical professor of neurologic surgery, George Washington University School of Medicine; with a foreword by Percival Bailey, M.D. 8°, cloth, 408 pp., with 128 illustrations. Chicago: Year Book Publishers, Incorporated, 1954. $12.00.

The author discusses cerebral disorders due to vasospasm, embolism, thrombosis and hemorrhage, as well as various types of brain disease. The preliminary chapters deal with the embryology of the intracranial vessels and the anatomy and physiology of the arteries, veins and sinuses of the brain. There are also chapters on inflammatory and collagenous diseases, blood dyscrasias, vitamin deficiencies and poisons, headache, diagnosis and treatment and general management of the patient. Extensive lists of references are appended to the chapters, with citations in the text. The book is well published and should be in all medical libraries and in collections on the subject.

Psychoanalytic Psychiatry and Psychology: Clinical and theoretical papers. Austen Riggs Center. Vol. 1. Edited by Robert P. Knight, M.D., and Cyrus R. Friedman, M.D. 8°, cloth, 391 pp., with illustrations. New York: International Universities Press, Incorporated, 1954. $6.00.

This collected volume contains 23 papers by 8 members of the staff of the Center. The material is divided into three main groups: clinical, clinicopsychologic and theoretical. There is a list of the writings of the Riggs staff, 1947 through 1951. Lists of references and indexes of names and subjects are appended to the papers. The volume should be in all collections on psychoanalysis.

Psychoanalysis and the Education of the Child. By Gerald H. J. Pearson, M.D., dean, Institute of the Philadelphia Association for Psychoanalysis. First edition. 8°, cloth, 356 pp. New York: W. W. Norton and Company, Incorporated, 1954. $5.00.

Dr. Pearson discusses his subject under three heads: psychoanalysis and the learning process; ego psychology and the education of the child; and psychoanalysis and the development of the moral sense.

Anatomy of the Human Body. By Henry Gray, F.R.S. Edited by Charles Mayo Goss, M.D., managing editor, *Anatomical Record*, and professor of anatomy, Louisiana State University School of Medicine. Twenty-sixth edition. 4°, 1480 pp., with 1202 illustrations, mostly in color. Philadelphia: Lea and Febiger, 1954. $16.00.

The major change made in this edition of a standard work has been a complete rewriting of the material on the peripheral system. For the first time lists of references have been appended to the chapters. There is a comprehensive index, and the publishing is excellent. The price is not excessive for this type of book. It should be in all collections on anatomy.

Proceedings of the Fourth International Congress of the International Society of Hematology, Mar de Plata, Argentina, September 20-27, 1952. Edited by F. Jimenez de Asua, William Dameshek and Sol Haberman. 4°, cloth, 473 pp., with illustrations. New York: Grune and Stratton, 1954. $10.00.

A large number of papers were read at the Congress and are presented in eight parts, as follows: neuroendocrinologic regulation of hematopoiesis; histochemistry and cellular ultrastructure; etiology and treatment of the leukemias; manifestations of radioactivity on hematopoietic organs and hemostasis; polycythemia; hemolytic disease and relation of immunology to hematology; hemorrhagic disturbances; and miscellaneous considerations. The volume is recommended for all medical libraries and to all physicians interested in hematology.

Legg–Calvé–Perthes Syndrome and Related Osteochondroses of Youth. By Charles Weer Goff, M.D., assistant clinical professor of orthopedic surgery, Yale University School of Medicine, attending orthopedic surgeon, Newington Home and Hospital for Crippled Children and St. Francis Hospital, Hartford, and visiting professor of physical anthropology, Hartford Seminary Foundation, Hartford, Connecticut; in association with Ned M. Shutkin, M.D., and Myrema R. Hersey, M.S. 4°, cloth, 332 pp., with 123 illustrations. Springfield, Illinois: Charles C Thomas, 1954. $10.75.

The author has based his monograph on an intensive study of crippled children and youths in the Newington Home and Hospital for Crippled Children. The text begins with a historical account followed by chapters on the syndrome and its genetics, on youth and development, constitutional aspects, the hip joint, x-ray determination, sociologic aspects and differential diagnosis. There is a long chapter on the other osteochondroses. The book covers the disease thoroughly. There is a bibliography of nearly 16 pages, as well as indexes of names and subjects. The book is recommended for all collections on bone and joint diseases.

Radiology for Medical Students. By Fred Jenner Hodges, M.D., professor of radiology and chairman, Department of Radiology, University of Michigan Medical School; Isadore Lampe, M.D., professor of radiology, University of Michigan Medical School; and John Floyd Holt, M.D., professor of radiology, University of Michigan Medical School. Second edition. 8°, cloth, 439 pp., with 106 plates. Chicago: Year Book Publishers, Incorporated, 1954. $8.00.

In this second edition a book first published in 1947 and reprinted twice has been revised and brought up to date. The text is divided into two parts on diagnostic and therapeutic radiology. The second part has been largely rewritten. The text is well illustrated and there is a good index.

Clinical Aspects of the Autonomic Nervous System. By L. A. Gillilan, Ph.D., M.D., associate professor of anatomy, Graduate School of Medicine, University of Pennsylvania. First edition. 8°, cloth, 316., with 42 illustrations. Boston: Little, Brown and Company, 1954. $6.50.

The author divides his text into two parts. The first deals with anatomy; the second discusses innervation of visceral organs and systems, with clinical applications, and is arranged by the systems of the body, with a chapter on visceral pain from the abdomen and pelvis. The literature has been reviewed, and there is a bibliography of 28 pages and a good index.

Meredith's Hygiene: A textbook for college students on physical, mental, and social health from personal and public aspects. By Arthur F. Davis, Ed.M., Dr. P.H., professor of physical education, Pennsylvania State University; and Warren H. Southworth, M.A., Dr. P.H., professor of health education, University of Wisconsin. Fifth edition. 8°, cloth, 906 pp., with 170 illustrations and 25 tables. New York: Blakiston Company, 1954. $6.00.

This new edition of a college textbook first published in 1926 has been revised and brought up to date. The material is well organized and thoroughly covers the subject of personal hygiene. A list of films and sources of supply are given in a supplement. There is a bibliography of nine pages and an index. The book could well serve as a ready reference tool in the medical library.

Textbook of Operative Gynaecology. By Wilfred Shaw, M.A. (Camb.) M.D., F.R.C.S. (Eng.), F.R.C.O.G. 4°, cloth, 444 pp.,with 382 illustrations. Edinburgh: E. and S. Livingstone, Limited, 1954. $19.00.

This beautiful book is a semiatlas. The chapters deal with the general procedures and the major gynecologic operations, which are shown step by step. Some of the procedures contravene Massachusetts law and cannot be circulated in that state. Lists of references are appended to the chapters, and there is a good index. The publishing is excellent. The book should be in all collections on the subject.

Principles of Internal Medicine. Edited by T. R. Harrison, Raymond D. Adams, Paul B. Beeson, William H. Resnick, George W. Thorn and M. M. Wintrobe. Second edition. 4°, cloth, 1790 pp., with 149 tables, 207 illustrations and 5 plates. New York: Blakiston Company, 1954. $16.00.

This revised edition of a book first published in 1950 is the joint work of 85 contributors, who are specialists in their particular fields. There are 311 subjects, and lists of references are appended to the subjects. The whole field of internal medicine, including the special systems, is covered. There is a comprehensive index of 87 pages. The book is recommended for all medical libraries.

Practice of Allergy. By Warren T. Vaughan, M.D., Richmond, Virginia. Revised by J. Harvey Black, M.D., Dallas, Texas. Third edition. 4°, cloth, 1164 pp., with 107 tables and 335 illustrations. St. Louis: C. V. Mosby Company, 1954. $21.00.

The first edition of this authoritative work was first published in 1939. In this new edition the text has been thoroughly revised and brought up to date. A chapter on pulmonary function has been added. There is a bibliography of 58 pages and a good index. The book is recommended for all medical libraries and to all persons interested in allergy.

Textbook of Medicine. By various authors. Edited by Sir John Conybeare, K.B.E., M.C., D.M. (Oxon.), F.R.C.P., physician to Guy's Hospital, London; and W. N. Mann, M.D., (Lond.), F.R.C.P., physician to Guy's Hospital, London. Eleventh edition. 8°, cloth, 904 pp., with 37 figures and 30 plates. Edinburgh: E. and S. Livingstone, Limited, 1954. $7.00.

This standard British textbook, first published in 1929, has been issued since then in 17 printings of 11 editions, speaking well for its soundness and popularity. The text is the joint work of 18 contributors and has been revised and brought up to date. The sections on psychologic medicine and the endocrine disorders have been extensively revised. New material includes the treatment of uremia and the nephrotic syndrome, collagen diseases and disease of the intervertebral disk, disorders of the blood and neurology. Three appendixes deal with examination for life insurance, the sulfonamides and the antibiotic drugs, and cortisone and ACTH. There is a good index, and the publishing is excellent.

Beyond the Germ Theory: The roles of deprivation and stress in health and disease. Edited by Iago Galdston. 8°, cloth, 182 pp., with illustrations. New York: Health Education Council, 1954. $4.00. (A New York Academy of Medicine book.)

The papers in this volume were presented at the twelfth annual Eastern States Health Education Conference. Eleven papers were read on deprivation and the stress syndrome. They are divided into four sections on dynamics, and nutritional, psychologic and social aspects. The text concludes with a list of references and notes, and there is a good index.

The Surgery of Pulmonary Tuberculosis. By James H. Forsee, M.D., colonel, chief, Surgical Services, Fitzsimons Army Hospital, Denver, Colorado. 8°, cloth, 208 pp., with 59 illustrations, 1 in color, 11 graphs and 46 tables. Philadelphia: Lea and Febiger, 1954. $6.50.

This monograph discusses in detail the place of surgery in the treatment of pulmonary tuberculosis. The text is divided into three parts — principles, operative procedure and experience. Lists of references are appended to the chapters; there is an index, and the publishing is excellent. The book should be in all collections on the subject.

Heart: A physiologic and clinical study of cardio-vascular diseases. By Aldo A. Luisada, M.D., associate professor of medicine and director, Division of Cardiology, University of Chicago School of Medicine, under a teaching grant of the National Heart Institute, United States Public Health Service, associate attending physician and chief of cardiac clinics, Mount Sinai Hospital of Chicago, and chief of service and cardiologist, La Rabida Jackson Park Sanitarium. With a foreword by Herrman L. Blumgart, physician-in-chief, Beth Israel Hospital, and professor of medicine, Harvard Medical School. Second edition. 4°, cloth, 680 pp., with 312 illustrations and 27 tables. Baltimore: Williams and Wilkins, 1954. $15.00.

This second edition of a book first published in 1948 has been revised and brought up to date. Chapters on cardiovascular syphilis, the prognosis of cardiovascular diseases and the social and legal aspects of heart disease have been added. The illustrations have been revised. The chapter on the history of cardiology presents a chronologic list from Botallo (1530) to Scherf (1949-1952). Lists of references are appended to the chapters, and there is a good index.

Arrest of Bleeding: Physiology, pharmacology, pathology. By Jacques Roskam, M.D., professor of internal medicine, University of Liège, Belgium. 8°, paper, 71 pp., with 21 figures. Springfield, Illinois: Charles C Thomas, 1954. (Publication No. 217, *American Lecture Series.*) $2.75.

The author discusses his subject in detail. There are special chapters on the prophylaxis and general treatment of hemorrhage and on the treatment of thrombosis. There is a list of 62 references.

NONCLINICAL NOTES

SIMMONS MEMORIAL LECTURE

Dr. John H. Dingle, professor of preventive medicine at Western Reserve University School of Medicine and director of the Armed Forces Epidemiological Board Commission on Acute Respiratory Diseases, gave the first James Stevens Simmons Memorial Lecture at Walter Reed Army Medical Center on April 21.

The lectureship, named for the late dean of the Harvard School of Public Health, will be given annually. Dr. Dingle spoke on "Respiratory-Disease Research and Military Preventive Medicine."

NOTICES

ANNOUNCEMENT

Dr. Samuel H. Epstein announces the removal of his office to 412 Beacon Street, Boston, for the practice of neurology.

MASSACHUSETTS ACADEMY OF GENERAL PRACTICE

A meeting of the Springfield Regional Chapter of the Massachusetts Academy of General Practice will be held at the Highland Hotel, Springfield, on Wednesday, May 25, at 6:30 p.m.

Dr. John W. Turner will discuss recent developments in radiology.

LABORATORY REFRESHER TRAINING COURSES

The schedule of the laboratory refresher training courses to be given by the Laboratory Branch of the Communicable Disease Center, Public Health Service, Department of Health, Education and Welfare, from July, 1955, to June, 1956, is available from the Communicable Disease Center, United States Public Health Service, P. O. Box 185, Chamblee, Georgia.

SOCIETY MEETINGS AND CONFERENCES

May. Sanitary Engineering Center Training Programs. Page 122, issue of July 15.

May 20–26. Consultation Clinics for Crippled Children in Massachusetts. Page 733, issue of April 28.

May 20. American College of Cardiology. Page 414, issue of March 10.

May 20. New England Rheumatism Society. Page 785, issue of May 5.

May 20–25. Lahey Clinic Lectures. Page 289, issue of March 17.

May 23–27. American Trudeau Society. Page 462, issue of March 17.

May 24. South End Medical Club. Page 828, issue of May 12.

May 25–June 15. Boston State Hospital. Psychiatry Seminars. Page 461, issue of March 17.

May 25. Massachusetts Academy of General Practice. Notice above.

May 26. Boston City Hospital House Officers' Lectures. Page 509, issue of March 24.

May 29–June 2. American Society of X-Ray Technicians. Page 553, issue of March 31.

June 1–4. American Proctologic Society. Page 648, issue of April 14.

June 1–5. American College of Chest Physicians. Page 553, issue of March 31.

June 5 and 6. American Board of Physical Medicine and Rehabilitation. Page 794, issue of November 4.

June 8–10. Woman's Auxiliary to the American Medical Association. Page 647, issue of April 14.

June 7–9. United States Public Health Service Medical-Officer Examinations. Page 509, issue of March 24.

June 10–12. American Electroencephalographic Society. Page 647, issue of April 14.

June 13–15. American Neurological Association. Page 689, issue of April 21.

June 13–17. European Rheumatology Congress. Page 742, issue of April 28.

June 21–25. National Association for the Prevention of Tuberculosis. Page 540, issue of March 25.

July, August and September. Beth Israel Hospital Course. Page 509, issue of March 24.

July 1–4. International College of Surgeons. Page 828, issue of May 12.

July 10–16. Congress of Nobel Prize Winners. Page 510, issue of March 24.

July 11–15. Symposium on Tuberculosis. Page 509, issue of March 24.

August 28–September 2. American Congress of Physical Medicine and Rehabilitation. Page 157, issue of January 27.

October 6–8. Academy of Psychosomatic Medicine. Page 509, issue of March 24.

October 22–26. American Heart Association. Scientific Session. Page 603, issue of April 7.

October 23–27. American Institute of Dental Medicine. Page 414, issue of March 10.

November 6–13. Second International Congress of Allergology. Page 157, issue of January 27.

November 19–21. American Association of Blood Banks. 8th Annual Meeting. Palmer House, Chicago, Illinois.

November 29–December 2. Interim Session of the American Medical Association. Boston.

CALENDAR FOR THE WEEK BEGINNING THURSDAY, MAY 26

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*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*8:45–9:45 a.m. Cardiac Grand Rounds. Jimmy Fund Building, Children's Medical Center, 35 Binney Street.

*9:00–10:00 a.m. Surgical Rounds. Sherman Auditorium, Beth Israel Hospital.

*9:00–10:00 a.m. Arthritis Grand Rounds. Robert Breck Brigham Hospital.

*9:00–10:00 a.m. Grand Rounds. Russell Room, House of the Good Samaritan.

*10:00–11:00 a.m. Combined Medical-Surgical Rounds. Sherman Auditorium, Beth Israel Hospital.

10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*11:00 a.m.–12:00 m. Thoracic Conference. Management and Rehabilitation of Chest Injuries. Drs. Dwight E. Harken and Harrison Black. Main Amphitheater, Peter Bent Brigham Hospital.

*11:00 a.m.–12:00 m. Medical Staff Conference. Sherman Auditorium, Beth Israel Hospital.

*11:00 a.m.–12:00 m. Pediatric Conference. Mount Auburn Hospital, Cambridge.

*11:00 a.m.–12:30 p.m. Hand Clinic (Physical Medicine and Rehabilitation Service). Boston City Hospital.

*12:00 m.–1:00 p.m. Studies in Calcium Metabolism. Dr. William H. Baker, Stearns Auditorium, New England Center Hospital.

*12:00 m.–1:00 p.m. Surgical Staff Conference. Sherman Auditorium, Beth Israel Hospital.

*12:15–1:15 p.m. Tumor Clinic. Dr. T. W. Botsford. Amphitheater, Peter Bent Brigham Hospital.

*7:00 p.m. Boston City Hospital House Officers' Lecture. Pathological Basis for the Treatment of Gastric Cancer, to Include End Results. Dr. Gordon McNeer. New Cheever Amphitheater, Dowling Building, Boston City Hospital.

FRIDAY, MAY 27

*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*9:30–11:30 a.m. Medical and Surgical Grand Rounds. Drs. George W. Thorn and Francis D. Moore. Main Amphitheater, Peter Bent Brigham Hospital.

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